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AMERICAN JOURNAL OF OPHTHALMOLOGY

Vol. 5 OCTOBER, 1922 No. 10

HOW TO KNOW THE BLOOD PRESSURE IN THE VESSELS OF THE RETINA.

A. P. MAGITOT, M.D.

PARIS.

The use of pulsation in the retinal vessels to determine when pressure made upon the eyeball causes the intraocular pressure to equal the blood pressure, has been worked out by Magitot and his associate Bailliart. The results of their studies are here given. The relation of intraocular and blood pressure to pulsation is explained; and that between the general blood pressure and the pressure in the retinal vessels is given. The technic, including the instrument by which pressure is made on the eyeball and the manner of using it, is given. The clinical applications worked out include recognition of hypertension in the spinal fluid, before the occurrence of choked disc, application to glaucoma, and angiosclerosis. For earlier work on this subject see p. 824.

Altho the phenomenon of retinal pulsation was already known, it is to Bailliart that is due the merit of having, in 1917, shown all the interest there might be in making use of this phenomenon for the physiologic and clinical study of the circulation of the blood in the eye, in a word, to observe "de visu," thru the ophthalmoscope, pulsations which in every other part of the organism we are obliged to feel, to listen to, or to register with different instruments.

The principle of the method consists in applying to an eye, the tension of which is known, pressures with a small instrument graduated in grams. By these means we provoke the appearance of the arterial pulse on the disc. At this precise moment, the pressure of the blood column is balanced by the pressure of the eye, and if it be possible for us to calculate this pressure in millimeters of mercury, we know the diastolic pressure of the retina.

It was with this end in view, that with Bailliart we undertook, in 1919, experimental studies to draw up a chart enabling us to estimate in millimeters of mercury the pressures exerted in grams on the eye. This subject was explained and developed by us at the International Congress of Washington last April. But in order to facilitate the use of the method by our colleagues, who have kindly interested themselves in the matter, my object in writing this article is to develop certain points which I was obliged to pass over during my oral communication, and on the other hand, to show what clinical interest every practitioner can draw from it.

PULSATION AND BLOOD PRESSURE.

Everyone knows that if the blood flowed in the artery with a constant pressure, there would be no pulsation. The arterial pulsation is due to the varying pressure of the blood column, difference between the two extreme cardiac phases, systolic and diastolic.

Normally the retinal arteries have a pulsation, and if this phenomenon is not perceptible with the ordinary methods of investigation, the reason is: First, the fact that the counterpressure exerted by the ocular tension on the arterial walls is not sufficient to balance the pressure of the blood column.

But if it happens that, the ophthalmotonous increasing, the tension rises to the same level as the blood pressure, the pulsations appear in all their amplitude. This elevation of the ophthalmotonus is realized when pressure is applied to the eyeball either with the finger or with Bailliart's pressure gauge. It occurs also in certain pathologic cases; and everyone knows the spontaneous pulsations which are seen to appear in some glaucomas. We

can draw from these facts the following conclusion: *The diastolic arterial pressure, i. e. the lowest, is normally higher than the ocular tension.*

For the veins, the question of the pulse is a little different. Its aspect is not similar to the arterial pulse. The arterial pulsation has the appearance of an abrupt, jerky movement, and this is easily understood when we remember its cardiac mechanism. The venous pulsation, on the contrary, takes the aspect of a rhythmic and slow sinking movement of the wall. Still, in certain cases, the venous pulse takes on the appearance of a piston like movement along the axis of the vessel.

Finally, whereas in a normal eye we never see spontaneous arterial pulsation, it often happens that we observe a *venous pulsation.* But when, as happens in 50% of the cases, this venous pulsation does not exist, it is always possible to provoke its appearance:

1. By pressing on the eye. This act increases the ocular tension. The venous pulse then appears, as does the arterial pulse when the ophthalmotonus balances the blood column.

2. By applying massage to the eye for a few minutes, or by pressing on it for a fairly long time with the finger. The result of this is to diminish the ocular tension. Therefore, when the venous pulse appears under these conditions, it is because the venous pressure was less than that of the ophthalmotonus.

From this we deduce the following physiologic principles:

1. *The arterial pressure is always higher than the ocular tension.*

2. *The venous pressure is not always so great as the ocular tension.*

We can, finally, lay down the following rule:

In order that pulsations may appear in the arteries or veins of the disc, it is necessary that the pressure exerted by the ocular tension on the wall of the vessels should balance the pressure of the blood column.

We have just spoken of pulsations. But let us suppose now that we press hard enough on the eye to drive the blood out of the arterial trunks, in other words raising the ocular tension to a higher level than the pressure of the liquid column. Then, at the instant when the last pulsation disappears, we can say that we have reached the limit of the greatest arterial pressure, i. e. the *systolic* pressure. This action may in fact be compared with that of the apparatus which compresses the humeral artery in the methods derived from Riva-Rocci's procedure.

Theoretically, it is always possible to know the systolic retinal pressure, and in Bailliart's method one should always endeavor to reach it by continuing to press with the instrument. But, practically, this is not always possible, and in cases of glaucoma, in which the tension is already very high, the pressure on the eyeball brings on violent pains or increases to such an extent the ophthalmotonus, that the disc appears in a fog. The knowledge of the systolic pressure, which is so important in physiology, is not, however, indispensable clinically, and the practitioner will find more profit in the knowledge of the diastolic arterial pressure. He should know that in a *normal eye:*

The *diastolic* arterial pressure of the retina is from 30 to 35 mm. Hg.

The *systolic* arterial pressure of the retina is from 70 to 80 mm. Hg.

But this is not all. He should compare this local pressure with the general pressure. In France, to estimate this pressure, we make use of a brace apparatus, such as Pachon's apparatus, or of instruments based on the auscultatory method, such as the Vaquez-Laubry apparatus. Like Bailliart, I give the preference to the latter, which, in a normal subject, gives the following figures:

Systolic brachial pressure, 140 mm. Hg.
Diastolic brachial pressure, 70 mm. Hg.

If we compare these figures with those of the arterial pressure in the retina, we get the following rule established by Bailliart:

a. ***In the case of a normal general pressure, the ratio between the local arterial pressure and the general pressure***

is as 0.45 *is to* 1. (*General pressure* =1; *Retinal pressure* = 0.45.)

b. *In the case of high general pressure, the ratio between the local arterial pressure and the general pressure is as* 0.50 *is to* 1.

This rule is very important to know, and we shall see that the variations of these ratios have a considerable value from a clinical point of view.

Let us consider again the venous circulation.

The systolic venous pressure has not the importance of the systolic arterial pressure. From what I have written above, it follows that the diastolic venous pressure is always near to the ocular tension; that in certain cases it does not reach the figures of the ophthalmotonus, whilst in certain others it goes beyond it slightly. We conclude from this that the average figure of the diastolic venous pressure is about 22 mm. Hg. This figure may, however, vary in the same subject according to whether he is fasting or has eaten, according to the muscular work, the effort, etc.; in a word, all the acts which set up a stasis in the large veins, including the act of pressing on the patient's neck to compress the jugular vein.

But suppose we press rather hard on the eye: we shall immediately see the venous pulsations cease and then the vessel will appear to shrink and grow white as if its walls became thicker as the contents were reduced to a small thread of blood. If we continue to press, it will soon be seen that, before the stoppage of all circulation, the current dissociates itself and takes on a granular aspect. It is exceptional in a normal subject to see the vein resist even a hard pressure and not become flattened, under the influence of the pressure.

TECHNIC.

1. It is indispensable *to take first of all the ocular tension of both eyes* after having instilled a few drops of holocain. This tension is carefully noted.

2. The examination may be made with the patient either sitting, or lying, but it is customary to place oneself under the ordinary clinical conditions, i. e. the patient seated in front of the observer. According to preference, either the direct or the inverted image may be used. Similarly, pressure may be applied by the operator himself on the eyeball with the left hand whilst the right holds the ophthalmoscope. But it is essential to have a sufficient magnification of the optic disc to catch the first pulsation. The direct image gives the desired enlargement, but if we desire to use the inverted image, we must make use of Gullstrand's small hand ophthalmoscope. Recourse should then be made to an assistant to apply the pressure to the eye, the left hand being occupied with the magnifying glass.

We will suppose that we make use of the erect image:

(a) To examine the right eye the operation is simple. The right hand holds the electric ophthalmoscope. The left holds the pressure gauge perfectly horizontal between the thumb and the forefinger. The rounded end of the rod is introduced between the external canthus and applied to the sclerotic, whilst the patient's gaze is directed slightly towards the nose. By means of the little and third fingers the hand should support itself on the temporal region and hold the apparatus firmly while waiting to begin the pressure on the eyeball. The figures marked on the rod of the apparatus should be turned towards the observer. (Fig. 1.)

The attention of the ophthalmologist should be *directed to the disc. It is there and not elsewhere that he must observe the blood flow.* It is in fact the only place where the pulsations are clearly visible, for it is the sole region in which the vessels are not covered with glial tissue and optic fibers.

The observer should also accustom himself from the start to distinguish rapidly the veins from the arteries, and then every beginner should train himself to note the difference between the arterial and the venous pulse.

(b) When the observer obtains perfect visibility of the vessels, he begins to press on the eyeball. As soon as he perceives a first pulsation either ar-

terial or venous, he must hold his hand perfectly still and, ceasing to look at the disc, direct the light of his ophthalmoscope on the scale to note the figure. Then he looks again at the disc and continues the pressure until the pulsations have ceased. He then notes the figure once more.

Most Important Remark. Any pressure on the eyeball causing a fall in the ophthalmotonus, it is indispensable not to press too slowly. Likewise, if a mistake has been made and it is considered necessary to take fresh measures, an interval of five minutes must be allowed so that the tension may become normal again.

When one has observed the first pulsation which gives the diastolic pressure, one continues rapidly to reach the systolic pressure. If pressure is applied too slowly the following phenomenon is observed:

The disc is seen to grow white, then the pulsations begin again. If the pressure is continued and slowly increased, the pulsations do not disappear. This is due to the fact that the *more one presses on the eyeball, the more the tension falls.* The figures obtained will be false for they cannot be utilized with those of the initial tension.

Every beginner should above all, practice studying first the diastolic pressure. It is the most important. When he has acquired a certain familiarity with it he will study the systolic pressure. But in principle one should practice noting the exact moment at which the first arterial or venous pulsation appears. For a certain time the estimation will be wrong; for the observer, not being sufficiently trained, will not cease his pressure until he has gone beyond the critical figure. But we may boldly affirm that a practitioner who knows how to use his ophthalmoscope will not take more than ten days to acquire the necessary skill.

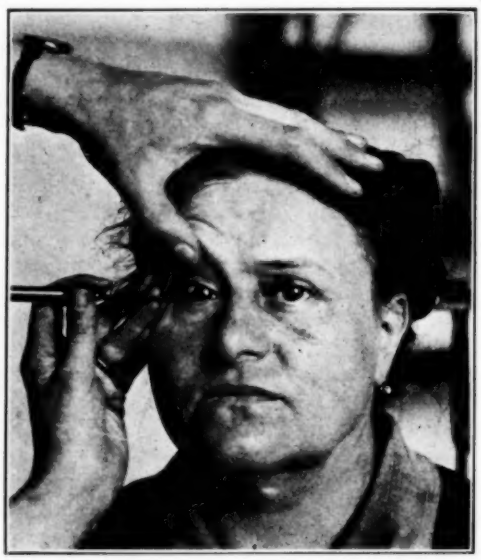

Fig. 1. Method of making pressure on the eye to induce pulsation of the retinal vessels.

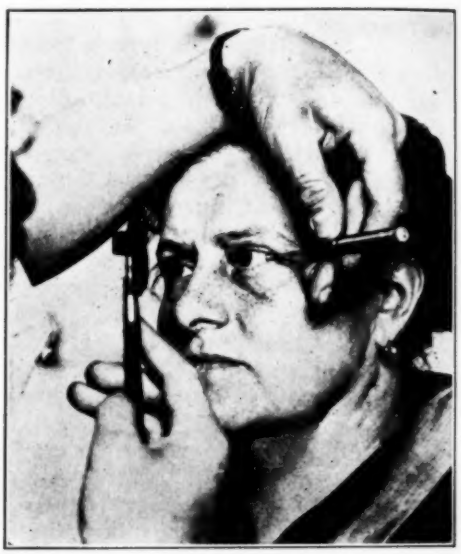

Fig. 2. Method of making pressure on the eye and seeing pulsation produced, if not able to use left hand for ophthalmoscope.

(c) The examination of the vessels of the left side necessitates a certain dexterity on the part of the observer. He is in fact obliged to hold the pressure gauge in the right hand and his ophthalmoscope in the left.

Dr. Bailliart, who was unable to become left handed, has recourse to the method indicated in figure 2.

I could add that for my part I practice the examination of the vascular pressure by utilizing Gullstrand's hand

ophthalmoscope, and the pressure on the eyes is applied by a nurse or an assistant. The assistant places himself behind the patient's head; he immobilizes the pressure gauge as soon as I see the pulsation appear, and it is he who reads the figure on the scale. When one is accustomed to the method, not more than one minute is usually required to know the diastolic and systolic pressures of the retina.

(d) Bailliart's pressure gauge,[1] is a simple apparatus of which two or three other models exist. The current model is that represented in figure 3. It has the advantage of being sensitive, but its scale is difficult to read, particularly in the dark.

A new model, actually under construction, has a dial on which the figures are much more easy to read.

These apparatuses are made to be used horizontally, so that their own weight may not affect the results. It is moreover very easy to verify their correctness by means of a pair of scales. The instrument is firmly held in a clamp (Fig. 5), and its lower extremity is placed in contact with one of the scales. It is sufficient then to place in the other scale the weight in grams to verify the exactness of the figures on the scale of the pressure gauge.

The spring which forms the principal part of the gauge is of hard steel, so that practically it insures the precision of the apparatus for a long time. But it sometimes happens that the rod is slightly out of true, producing abnormal friction. Similarly, in the long run, dust may penetrate between the two parts which fit into each other. All that is necessary is to have the apparatus taken to pieces by a mechanic and to have it trued on a lathe, if required.

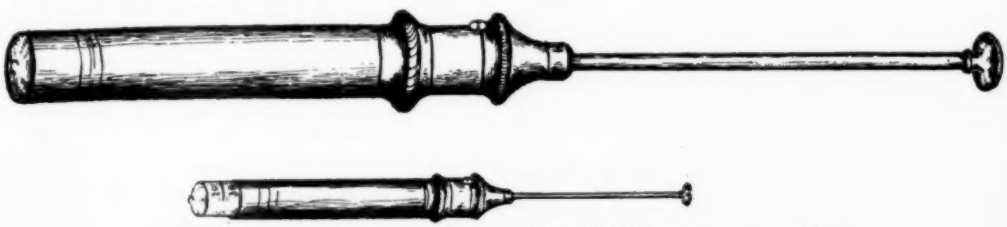

Fig. 3. Bailliart's pressure gauge for making definite pressure on eyeball.

CLINICAL APPLICATIONS.

It is extremely important to be imbued with the idea that the figure of the local blood pressure has a clinical value, only if it can be compared:

(a) with the figure of the ocular tension.

(b) with the figure of the general pressure (systolic and diastolic).

1. The ocular tension will be taken with Schiötz's tonometer. This tonometer is indeed the one which was used to draw up the chart herewith, which enables us with two readings to convert into mercury millimeters the figure indicated by the gauge. The reader will easily perceive that the abscissas are the markings in grams whereas the ordinates show the markings in millimeters of mercury.

Here is an example: Suppose an eye, the initial tension of which (before any action is taken) is 20 mm. Hg., and in which the first arterial pulsation appears under a pressure of 30 grams. Take the chart and follow the curve which begins at the figure 20 mm. Hg. Follow it until we reach the column headed by the figure 30, and look now for the level we have reached in mercury millimeters. We find 35 mercury millimeters. It is the figure of the retinal blood pressure. (Fig. 4.)

As this figure depends on that indicated on the tonometer, it will be easily understood that it is important to have a perfectly exact Schiötz to-

(1) Bailliart's pressure gauge is made by the firm of Boulitte, Paris, Rue Bobillot, 16. The model with a dial is made by the firm of Pirard & Coeurdevache, Rue Blainville No. 7, Paris.

nometer. This is not always the case with those sold in the shops. Those we made use of for drawing up the chart were tonometers which had been verified in the course of manometric experiments.[1]

2. Knowing the ocular tension and the local blood pressure, it is finally necessary to know the general blood pressure. For this, use should be made of one of the modern apparatuses with armlet placed on the arm and, to insure further precision, the auscultatory method should be used.

It is the comparison between the figures of the local and general blood pressure which alone has a clinical value and the following rule which constitutes the *normal formula* should be remembered:

(a) The normal arterial general pressure and local arterial pressure are to each other as 0.45 is to 1. Example:
General diastolic pressure...60 mm. Hg.
Local diastolic pressure.....35 mm. Hg.

(b) The high general arterial pressure and the local arterial pressure are to each other as 0.50 is to 1. Example:
General diastolic pressure..100 mm. Hg.
Local diastolic pressure.... 50 mm. Hg.

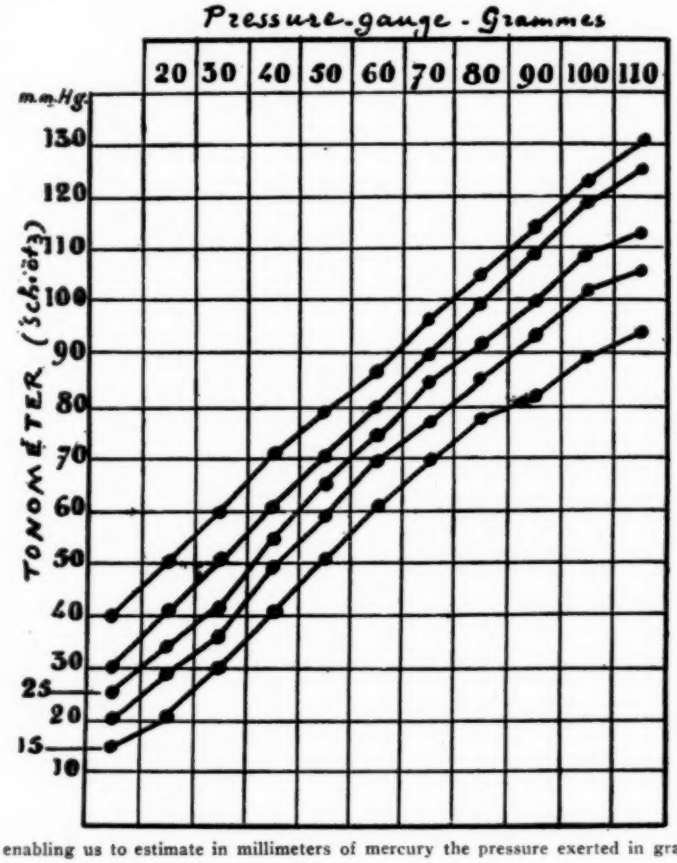

Fig. 4. Chart enabling us to estimate in millimeters of mercury the pressure exerted in grams on the eye.

Starting from these normal formulae we will review a few pathologic formulae:

A. Choked Disc.

The formula is as follows:

The *general* blood pressure:
Diastolic .....normal
Systolic .....normal

The *local* blood pressure:
Diastolic .....high
Systolic .....often normal

(1) The firm of Guilbert, Opticien, Rue de l'Odéon, Paris, sells at present tonometers which have been tested and marked at the Lariboisere Hospital.

The study of a certain number of clinical cases has moreover shown that every time this formula has been met with, there was a hypertension of the spinal fluid even if there was no choked disc. It is therefore clinically possible now to discover an important pathologic phenomenon before the appearance of the objective sign which is a consequence of it.

Here is an instance: A man who had been wounded in the war, and who showed some symptoms of Jackson's epilepsy, complained of pains in the head. There was no choked disc but I found an ocular tension of 30 mm. Hg., a general diastolic pressure of 70 mm. Hg. and systolic of 135 mm. Hg. The local diastolic pressure was 70. mm. Hg. The normal formula (as 0.50 is to 1), no longer existed since the two diastolic pressures, local and general, were identical.

I diagnosed great hypertension of the spinal fluid. Puncture in the lumbar region justified this diagnosis and by means of radiography a small intracerebral projectile was discovered.

B. Angiosclerosis of the Retina.

General pressure:

Diastolic .....high, normal, low
Systolic .....high, normal, low

Local pressure:

Diastolic .....low
Systolic .....low

The arterial pulse is besides often feeble. Sometimes it is located in a segment of the arterial trunk.

Example: A patient complains of visual fog. The vessels, arteries and veins, appear normal, but the general diastolic pressure is 120 mm. Hg. The local pressure is diastolic 45 and systolic 75.

If we compare the difference between the diastolic pressures, we no longer find the proportion of 0.50 to 1, but of 0.38 to 1. Moreover, it must be noted that the local differential pressure is diminished, i. e. the difference between 45 and 75 is indeed much less than the normal difference (which is 30–75). This formula appears before the hemorrhagic lesions. It is a precocious symptom which enables us to diagnose vascular lesions when the patient, while still possessing normal sight, complains of passing obnubilations, and of myodesopsia. A very similar formula is found in retinitis gravidica and albuminurica.

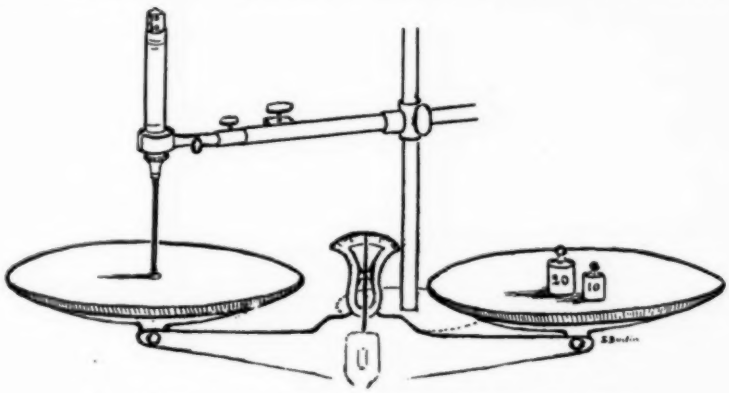

Fig. 5. An easy way to test accuracy of the pressure gauge.

C. Glaucomas.

In glaucoma in general we find the following formula:

General arterial pressure:

Diastolic .....normal or high
Systolic .....normal or high

Local arterial pressure:

Diastolic .....high
Systolic .....high
Local *venous* pressure.....very high

In 57 cases of glaucoma Bailliart found eleven times the spontaneous arterial pulse and seven times the spontaneous venous pulse. This indicates that in 19% of the cases, the arterial diastolic pressure was counterbalanced

by the ocular tension, and that in 13% the venous pressure was also very near to the ocular tension. As in these 57 observations, there was an average tension of 55 mm. Hg., it will be seen by that how great the local blood pressure is in glaucoma. This agrees very well with the idea I hold, that glaucomas are due to an inflammation of the choroid and not to an obstacle in the flow of the aqueous humour.

As to the local systolic pressure, it is often very difficult to know its exact figure. In an eyeball, the tension of which is already high, a pressure with the dynamometer clouds the vitreous humor and causes the visibility of the disc to disappear. Still, attentive study of certain cases has enabled us to find out:

That the ocular tension followed the local systolic pressure in a parallel manner.

That this local systolic pressure was relatively less high than the local diastolic pressure.

It is to the elevation of this arterial diastolic pressure that is due the appearance of the spontaneous pulse. The conclusion to be drawn is that the observation of the *spontaneous arterial pulse* should be considered as an alarm signal.

When, finally, we find ourselves in the presence of ocular disturbances rendering research for pulsations impossible, we should remember that every time that the ocular tension is superior to half the humeral pressure, the circulation of the retina is unquestionably disturbed.

As for the venous pressure, it is very high, since it sometimes reaches the figure of the ocular tension. It should be added that the venous circulation seems to be affected solely in the eyeball. In fact we see none of the ophthalmoscopic symptoms (tortuousness, edemas, hemorrhages) which indicate a difficulty of the circulation as in thrombosis of the orbital veins. Moreover, we know that, as soon as the veins have left the eyeball, they dilate; it is what takes place in the case of episcleral veins. The principal cause of this dilation is the pressure of the liquid column which is no longer counterbalanced by the ocular tension.

D. There are other affections in which the local blood pressure has been examined, but the cases studied are not yet sufficiently numerous to enable us to deduce a formula from them. It is thus that I have observed a case of posthemorrhagic blindness, cases of traumatic myopia, and that Bollack has observed a case of quinin blindness. But the method is still too recent to permit of yielding all we have a right to expect from it. It is for this reason that I have written this article, in order to interest my colleagues in a new technic which, from the results already obtained, seems to deserve to take its place in the current practice of ophthalmology.

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EPIDEMIC ENCEPHALITIS FROM THE STANDPOINT OF THE OPHTHALMOLOGIST.

GEORGE FRANKLIN LIBBY, M.D., OPH.D.

DENVER, COLORADO.

The recent history and general characteristics of this disease are described. Five cases are reported, in two of which there was inflammation and swelling of the optic nerve head. The importance of the disease is pointed out and the fact that it lies in the domain of the ophthalmologist. Read before the American Ophthalmological Society, May, 1922.

It is important for the ophthalmologist to have a working knowledge of epidemic encephalitis, because he may be the first physician consulted by the patient, or his counsel may be sought by the internist or neurologist. The ocular disturbances are often of rapid onset and sometimes of a very distressing character, and may be the first or even the only symptoms noticed.

Happ and Mason[1] have made a thoro clinical study based on 81 cases of this disease, observed in hospital wards and followed up after discharge. They refer to 119 publications on the subject.

The recent epidemic of encephalitis was first noticed in Austria and Northern France, during the winter of 1916-17. The following winter it appeared in Paris. It was soon recognized in other parts of Europe and in England; and by another year, probably aided by extensive troop movements, it had spread over the world, becoming a great pandemic.

The association of epidemic encephalitis with epidemic influenza, noticed in the last few years, has also been observed for four centuries; sometimes one disease preceded the other, again the two diseases occurred simultaneously. The lesions of the nervous system are many, diverse and widespread, hence the variety of descriptive adjectives coupled to encephalitis. The virus lingers for a long time in the central nervous system. As one area is recovering, another may be attacked, greatly prolonging the period of disability.

The first symptoms are likely to be those common to the initial stage of an acute infectious disease, such as fever, headache, vomiting, irritability and restlessness. The young may have delirium or convulsions. Later, there develop drowsiness, coma or insomnia, diplopia, ptosis or other ocular disturbances, tremors, muscular jerkings, spasticity, general weakness, mask like face, and various psychic disorders, especially during convalescence. Sometimes sleeplessness at night alternates with deep sleep by day.

The duration of the lethargic state for weeks or months gave rise to the first designation of this disease as "encephalitis lethargica." However, in some cases there is no lethargy. All things considered, probably "epidemic encephalitis" is the best classification for this disease.

Headache persisting for weeks or months may be wholly dependent on encephalitis, and not necessarily a sign of brain tumor or abscess, even in the presence of papilledema. The headache of tuberculous meningitis is of a different character, and this disease is usually rapidly fatal.

Of the ocular symptoms, diplopia is frequently the first indication of involvement of the nervous system, and may be the first sign of encephalitis. The palsies of the eye muscles are characteristically transient and variable. Ptosis is frequent, often bilateral, seldom complete. Weakness or even loss of convergence is common; insufficiency of accommodation also occurs. Nystagmus of various types is present in a large number of cases. Pupillary inequalities and irregularities are seen. Optic neuritis is occasionally observed, either in a mild or a severe form, and in one or both eyes.

Holden[2] makes the observation that ophthalmologists have noted more cases of paresis of accommodation and diagnosed paresis of motor muscles of the eyeball more accurately, than is done by hospital groups, altho he ac-

knowledges the statistical value of such reports and draws valuable data therefrom. Eye symptoms noted in 100 consecutive cases of epidemic encephalitis, at the Mount Sinai Hospital, gave the following percentages: Blurring of the optic discs, 4. Papilledema, 1. Ptosis of both eyes, 45; of one eye only, 11. Paresis or paralysis of the various motor muscles of the globe sufficient to cause diplopia, 55, of which 44 were of the external recti. Nystagmus, 32. Irregular pupils, 15; unequal, 20; sluggish or absent light reflex, 35, in 13 of which the convergence or accommodation was also sluggish. Weakness of accommodation in both eyes, 1. Weakness of some or all of the muscles supplied by the facial nerve: both sides in 24, one side only in 49.

Holden believes that true papilledema is a very rare symptom of encephalitis, a concomitant of unusual complications, and usually the result of increased intracranial pressure. He points out that from the widespread inflammatory changes in the brain, muscular disabilities varying from weakness to paralysis must occur. Diplopia usually disappears early, ptosis later, and pupillary anomalies and weakness of accommodation last of all. He thinks that during an epidemic, encephalitis may be diagnosed from the ocular symptoms alone if lues is eliminated.

The microscope shows no essential difference between the vascular and perivascular changes of syphilis and those of encephalitis. This would account for the similarity of symptoms in the two diseases. Dickinson[3] speculates on the frequency with which mild cases of encephalitis have been diagnosed as syphilis of the central nervous system and treated as such, and their recovery attributed to mercury and the arsenol compounds. A very important point in differentiation is the duration of the muscular palsies. In syphilis the lesion is generally of long standing before it yields to treatment, while in encephalitis the paralysis is fleeting, and may shift from one muscle to another.

Spaeth[4] observed five cases of "encephalitis lethargica," all of which showed various ocular symptoms. One case had binocular optic neuritis of a mild type, another had partial corneal anesthesia in one eye and almost complete anesthesia in the other, and all five cases had muscular involvement. All were more or less acutely ill, the longest period of disability being 3 months. Spaeth believes that "great care must be taken as to treatment during the acute, irritant stages of the disease." This also applies to a rather prolonged period following recovery, to prevent possible relapse.

Taylor[5] reports six cases in which diplopia or ptosis, or both these conditions, were amongst the earliest symptoms of which the patients complained. The general clinical picture was that of encephalitis. One case showed double ptosis, divergence of one eye, weakness of all eye movements and nystagmus to right and left, with normal pupillary reactions. Some of Taylor's cases had the aspect of paralysis agitans without tremor, and others a mask like face, after the disease had run its course. Only two of his cases made a complete recovery.

Monro[6] describes seven cases, one of which had well marked optic neuritis, with hemorrhages. He gives a good description of the cardinal symptoms of epidemic encephalitis and alludes to its special tendency to attack the pons and midbrain, causing edema, hemorrhage and degenerative changes.

Pickard[7] relates five cases, all of which showed diplopia and paresis of accommodation. Four of the cases had optic nerve disturbances. He thinks that mild cases of encephalitis may be commoner than is thought. The ocular symptoms establish the diagnosis in such cases.

Haynes[8] gives a good description of the disease with the exception of the statement that the fundus oculi is normal, and quotes Farquhar Buzzard's observation that an inflammation of the encephalon can produce as many symptoms as there are functions of the brain. He also outlines the treatment.

Commenting on the few references to changes in the fundus oculi or in the visual function, that he had been able to find in the literature on encephalitis, Symonds[9] reports four cases of bilateral optic neuritis which he observed at American hospitals which he visited.

Patton[10] considers encephalitis from the ophthalmic viewpoint, carefully reporting three cases. The discussion of his paper brings out interesting features. Reference is made to the statement of Morax, that when ocular symptoms are wholly absent, the propriety of classifying cases as "lethargic encephalitis," is questionable. Patton calls attention to the importance of determining the presence or absence of encephalitis, in the presence of unexplained diplopia, ptosis or irregular pupillary reaction.

Hogue[11] reports the incidence of the disease and the mortality in Wisconsin, and records four cases with the usual eye symptoms. He details the frequency of the various ocular disturbances, and the order of their disappearance. Only two of the cases showed lethargy.

Waardenburg[12] describes two cases studied by himself, and compares them with the cases published in Holland, and reviews the accounts of this disease as given in other countries. The general symptoms are first outlined and the ocular manifestations, motor, visual and pupillary, are taken up in detail. Charts are given indicating fields of fixation as well as fields of vision. He also calls attention to the importance of the ocular symptoms in determining a diagnosis of encephalitis.

Griffith[13] elaborates his earlier study of acute nonsuppurative encephalitis, particularly as it affects the cerebellum, and as it is manifested in children. He has collected records of thirty-one cases, in addition to four reported by himself. They mainly occurred independently of any epidemic, often closely following acute infectious diseases, and could not be attributed to an encephalitis of any specific microbic nature, altho that relationship was possible. He considers the prognosis good as to survival, but that complete recovery is uncertain.

Cadwalader[14] points out the practical impossibility of separating the lesions of acute anterior poliomyelitis from those of epidemic encephalitis, under microscopic study. This suggests the naturally close resemblance of the clinical phenomena of these two diseases. Epidemic poliomyelitis occurs mostly in summer and chiefly among children, epidemic encephalitis usually occurring in the winter months and largely among adults. Lethargy is not always present in the latter disease, and it occasionally occurs in the former affection. The identity of the microscopic findings in some cases of epidemic encephalitis and in early cases of multiple sclerosis explains the resemblance of the manifestations of these diseases.

He calls attention to the rare but definite involvement of the spinal cord in epidemic encephalitis; and refers to a case of binocular blindness following optic neuritis.

Ophthalmic Literature[15] has a lengthy review which covers most of the important papers dealing particularly with the eye symptoms of encephalitis. Under the general title of Ocular Manifestations of Encephalitis[16], the European journals deal with this subject along the same lines already laid down; while Bollack[17] deals with nystagmus and Demole[18] with internal ophthalmoplegia, in this disease.

The following illustrative cases are taken from the practice of the writer, except case 4, which is kindly furnished by Dr. W. C. Finnoff.

Case 1. On Sept. 28, 1921, a man of thirty-one was taken with severe headache, delirium which lasted for a week, and fever which rose to 104° F. No diplopia was noticed. At the end of a month, when the eyes were first examined, the right pupil was larger than the left (but not dilated ad maximum) and the papillae were slightly hyperemic, the left disc being blurred at the nasal margin. There was one degree of right hyperphoria, the accommodation of the left eye was 1.25 D. weaker than that of the right, and there was marked asthenopia. A week later a "nervous break-down" occurred. Recovery from the acuteness of this at-

tack was followed by blurring of vision, marked dizziness and early fatigue of the accommodation, which had fallen off 0.50 D. more in the left eye. The pupils were now equal and the fundi were normal. The patient's nerve-tone remained very poor.

Case 2. On Nov. 19, 1919, a boy of fourteen years who had had fever, pharyngo-tonsillitis and general malaise for three days, suddenly developed bilateral mydriasis and marked ptosis of the left upper lid. Twelve hours later the pupils were normal as to size and reactions, and the ptosis was reduced one-half. Four days later the ptosis had disappeared. Soon after this the right pupil dilated once, for a few hours only. The ptosis returned for brief periods when very tired. The slowness of recovery, long after the throat was well, the lethargy and the ocular findings led to a diagnosis of a mild attack of epidemic encephalitis, as in the following case.

Case 3. A man of fifty-six presented himself on Nov. 22, 1919, stating that a week before he had vomited several times, diplopia appeared the next day, and severe frontal headache came on the second day following. He had never had double vision before. Examination showed twenty degrees of left hypertropia, the left eye turning 2 1/2 mm. above the horizontal meridian, and there was ptosis of the left upper lid. Otherwise the eyes were normal except for errors of refraction. The man was noticeably lethargic for nearly two weeks; after which the lethargy, diplopia and ptosis promptly disappeared.

Case 4. Three weeks before, a man of forty-six had suddenly developed severe pain in the head, and fever quickly followed. When the temperature was first taken it was 103°. Physical examination revealed a left sided pneumonia. In a few days he became lethargic and remained so for 4 or 5 days. As pneumonia was thought to be the cause of the constitutional symptoms, no neurologic examination was made, and the signs of lethargic encephalitis were not looked for. After recovering from the lethargy, the patient noticed that his vision was blurred and that he had diplopia; also, that he was confused mentally at times. His physician observed that the lids did not close entirely during sleep, and noted a condition of the eyes which he thought was lateral nystagmus. The pneumonia cleared in ten days but was followed by a pulse of only 29 per minute. The patient was very weak and dizzy most of the time. The vision continued to blur and diplopia was noticed; and it was for these conditions that an ophthalmologist was called on July 5, 1921. R. V. 20/20, L. V. 20/20. With his former reading correction he could read with either eye separately, but could not read with both. Slight diplopia. Muscles: Dist., left hypophoria 2.5 cr., esophoria 4 cr. Near, left hypophoria, 6 cr., increased in upper field. Confused mentally, so that the full error in all fields could not be measured. L. eye lags in upward and outward movements. Esophoria, 20 cr. Accommodation retarded. R. disc red, swollen 0.5 to 1 D., edges indistinct, veins engorged and slightly tortuous, arteries slightly contracted. L. neuroretinitis, disc swollen 2 D., arteries contracted, veins markedly engorged and tortuous, and small hemorrhages and exudates on surface of disc. July 10, R. V. 20/20 plus, L. V. 20/30 partly. L. hypophoria, esophoria. July 15—Gaining in strength. Only two short periods of blurred vision. Near: vertical balance, esophoria 6 cr. Slight improvement of optic disc. July 22—Feels much better, and does not become confused or lethargic. Eyes comfortable. V. 20/20 plus in each. Muscle balance good except 6 centrads of esophoria for near. R. disc less congested and less obscured at margins. L. disc swollen 1 D., its exudates less and its hemorrhages absorbing. Fields of both eyes normal by arc perimeter and by Peters' campimeter. July 29—Weak and dizzy, but clear mentally. V. 20/15. Near: vertical balance, and 0.5 cr. of esophoria. Slight blurring of upper nasal quadrant of R. disc, remainder normal. L. disc swollen only 0.5 D., margins still blurred, two flame shaped hemorrhages 1/6 d.d.

in upper half of disc, near margin. Aug. 6—Vertical and lateral balance for distance. L. disc decidedly improved, no swelling, hemorrhages absorbing. Dec. 19—Patient reports that he is well but not so strong as formerly.

CASE 5. On April 15, 1921, a man of twenty-two sought relief thru glasses from occasional "filmy vision" in the previous month, and headache, from which he had suffered for ten days. The headache was of a stabbing character, was in the lower occipital region, and occurred several times a day. There was a mild neuroretinitis in the right eye, while this condition was pronounced in the left, without hemorrhage. R. V. 5/4 partly, L. V. 5/10 partly. There was no central color scotoma. The patient was referred for examination of the nasal accessory sinuses, the tonsils, teeth, blood, spinal fluid, kidneys and other important organs. Thoro physical examination resulted in negative findings only. Slight tremor of the hands was noticed in 1919. There had been a moderate influenza in the fall of 1918; good recovery. In Aug. 1919, he received a blow on the head; not rendered unconscious. Radiographs showed "lack of the posterior clinoid process; also a small calcified area above the tentorium, which appears to be the center of a tumor mass." On Apr. 18 the left disc was choked, and the vision was down to 5/15. On the following day Dr. Robert Levy opened the left antrum, ethmoid and sphenoid, and did a double tonsillectomy. He found the ethmoid soft, the sphenoid and antrum normal, and that the small, submerged tonsils contained a scanty mucopurulent secretion. A week later the left disc was less swollen, and the vision had risen to 5/10 partly. The vision of the left eye dropped to 5/22 in the following eight days. May 2, 1921, the right disc was swollen 5.50 D. and the left 7.50 D. Both blind spots were much enlarged, especially the left. The lower field of vision in the left eye was largely lost. There were no retinal hemorrhages. Two days later spinal fluid found negative to all tests, as was the blood to the Wassermann test.

On May 6, Dr. H. T. Pershing began a neurologic study of the case, and ordered prolonged rest in bed, and prescribed potassium iodid and mercury. By May 31, the neuroretinitis had subsided in the right eye, and largely so in the left, with but one diopter of swelling. Vision was R. 5/4 partly, L. 5/22. On June 20, the right nerve protruded forward 4 D., the margin was clearly defined, and the vessels dipped over the edge. The left disc was swollen 2 D., and there were new vessels and minute hemorrhages on its lower nasal quadrant.

Sept. 10—R. V. 5/12 partly, L. V. 1/60. From this time the edema of the optic discs generally subsided, with occasional remissions, and the vision of the right eye gradually failed. The spinal fluid pressure was 400 mm. of water on Oct. 4, and 330 on Oct. 15. Vision improved slightly after the first withdrawal of the fluid, but the second had no such result. On Dec. 14, 1921, there was complete optic atrophy, loss of pupillary reflexes except to direct sunlight, and the vision light perception only in the left eye and possibly 1/60 in the right eye. The patient was still confined to the house; tho rather weak, was clear mentally and would soon go outdoors. In April, 1922, R. V. = 8/200, and the mental and physical condition was excellent.

The following note is from Dr. H. T. Pershing, who collaborated from the neurologic standpoint.

"My reasons for thinking this is a case of encephalitis are as follows:

"1. Meningitis is excluded by the infrequency and short duration of the headaches, the absence of vomiting, fever, delirium and of all cranial nerve affections except the optic neuritis, and the normal condition of the spinal fluid. The differential diagnosis is really between encephalitis and tumor.

"Against tumor are the following:

"1. The rapid onset: from the first symptom of which we are sure, until headache was severe and vision distinctly affected, was not more than a week or at most ten days.

"2. The absence of vomiting and of slow pulse.

"3. The entire absence at all times of any other cranial nerve symptom than the optic neuritis.

"4. The normal condition at all times of gait, station, voluntary motions and the reflexes, especially the knee jerks, heel jerk and plantar reflex.

"5. The infrequency of headache after the first onset of the illness. It occurred only as an immediate result of the spinal punctures.

"6. Absence of such progress in the development of symptoms as we generally see in tumor.

"In favor of encephalitis is the fact that the disease had been occurring in Denver and Colorado since the winter of 1918-19, and also that it can have a selective action on a limited portion of the brain. The optic nerve is really a part of the brain."

COMMENT

As will be observed, these five cases ran the gamut of ocular symptoms, from transient muscular palsies to destructive optic neuritis. In some the general manifestations were severe, in others mild. Cases 4 and 5 illustrate an involvement of the optic nerve not suspected earlier in the epidemic, but which the nature of the disease should lead us to expect.

The experience of the past four years justifies the conclusion that epidemic encephalitis is a disease of great importance, often of marked severity, and with a tendency to a prolonged course and to relapses. It so commonly attacks the eye, that a diagnosis of encephalitis in the absence of ocular manifestations, may well be doubted. On the other hand, it is possible to diagnose the disease from the eye symptoms alone, if syphilis is excluded. In mild cases there may be no symptoms complained of except the ocular disturbances. As no function of the brain is immune in encephalitis, so the various functions of the eye seem liable to attack. And as the virus of this dread disease shows its ill effects upon the general nervous system for years, so we may expect the eyes to show persistent if not permanent damage to the structures that were seriously affected. We must be careful, later on, not to confound these pathologic changes with those of nerve syphilis.

Epidemic encephalitis seems to lie in the domain of the ophthalmologist quite as much as in that of the neurologist or internist. However, the ideal attitude in the diagnosis and treatment of this as of all diseases, is one of cordial cooperation.

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SYMPATHETIC IRIDOCYCLITIS.

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A case is here reported following perforating injury in a man of 67, the eye being removed at the end of eleven weeks after the injury, five weeks after evidences of involvement of the second eye. Fair recovery followed extraction of infected teeth and treatment. Pathologic examination of the exciting eye by Dr. William C. Finnoff of Denver. The essential character of the disease is discussed in detail. Reviewing theories of pathogenesis heretofore put forward, the view is favored, that the condition is not specific, but may be brought about by infection from foci previously established within the body. Read before the California State Medical Association, May, 1922.

Mr. H., aged 67 years, received a perforating injury to the right eye, six weeks previous to coming under observation. Sight failed immediately following the injury. Owing to the patient being in the mountains many miles from medical attention, home remedies were the only measures instituted. The eye continued inflamed and at intervals more sensitive than others. Sight in the left eye had been failing for two years preceding the injury. Recently this eye became slightly red.

Examination showed the right eye to be absolutely blind, markedly injected, mushy, and very sensitive to touch. The degenerative changes incident to an eye becoming phthisical were present. There was evidence of a perforating wound at the nasal corneo-scleral juncture. The left eye showed a vision of 6/60. Very slight ciliary injection, iris responded promptly but irregularly and not fully to light stimulus. On the anterior lens capsule were two very minute pigment spots. The lens showed peripheral shoots and a very hazy nucleus.

Immediate enucleation of the injured eye was advised. The patient did not accept the advice at this time, but returned five weeks later (eleven weeks after the original injury). He complained that the sight of the left eye suddenly failed a few days previous. Moderately severe neuralgic pains radiating up over the left brow. The eye presented marked ciliary and conjunctival injection, ciliary tenderness, and a very hazy cornea. Deep in the cornea were several small mutton fat spots. Descemet's membrane showed numerous fine pigment deposits. The anterior chamber was shallow. The pupil irregularly round. The iris nonresponsive to light and bound down to the anterior capsule. The texture of the iris appeared disturbed, and at the pupillary border to the temporal side were two, slightly yellowish, dark, pin point nodules. Light field good. Vision equaled hand movements at two feet.

The offending eye was promptly removed, atropin, purges, sweats, and sodium salicylat were administered. Owing to the difficulty of controlling the patient, being ambulatory, these measures were carried out very indifferently. During the succeeding 18 days there was only moderate improve

ment. Temperature observation every three hours for five days, was normal, Wassermann, tuberculosis and gonorrhea reactions were negative. Radiographs of his teeth showed extensive disease in the form of dental caries, apical abscesses, pyorrhea, and alveolar necrosis. His mouth was thoroly cleaned up. Within twenty days the left eye showed definite improvement, vision equaled 1/60. At the end of two months, the eye, practically quiet, had a vision of 5/60. It is now 15 months since the attack; during this period there has been no recurrent outbreak. The lens has become more opaque and swollen; V. —4 = 6/15.

The question of removing the cataractous lens is a problem for the future.

The following excellent notes from Dr. Finnoff's careful study of the offending eye confirms pathologically the clinical diagnosis of sympathetic disease.

Microscopic Appearances. The corneal epithelium varies in thickness, and is thinned in the greater part, to about 3 layers of cells (probably postmortem artifact). Bowman's membrane is intract thruout its whole extent, is normal in thickness, but is wavy. The substantia propria varies in thickness, due to edema of the lamellar fibers. The greatest swelling is near the center of the cornea. The corneal cells have undergone cloudy swelling, and the intralamellar spaces have been separated by fluid. The irregular thickening of the substantia by edema, accounts for the wavy appearance of Bowman's membrane and a folding of Descemet's membrane. Near the limbus, on either side, the cornea thins down to almost its normal diameter. At the limbus, on the side which was wounded, Bowman's membrane dips backward and is covered. In front of this, under the epithelium, there is a thin layer of granulation tissue, in which there are several new formed pigmented epithelial cells. Some of the cells have broken down, and the pigment granules are seen scattered thruout the new formed tissue. In the substantia, on the wounded side, several blood vessels have wandered in between the lamellæ. For a distance of about 2 mm., Descemet's membrane is thrown into several sharp folds.

Only an occasional endothelial cell is seen on the posterior surface of the cornea. The corneoscleral lamellæ, in the region of the scleral spur, on the injured side of the eye, have been ruptured by the trauma, and the wound is filled with polymorphonuclear leucocytes, lymphocytes, plasma cells, degenerated pigment cells, pigment granules, and fibroblasts. The meshwork of the filtration angle on this side has been entirely destroyed. The base of the iris and sinus (Salzmann) of the ciliary body have been destroyed, and in this area, a mass of inflammatory cells, new formed capillaries, brokendown pigment cells, and pigment granules are found. The filtration angle of the opposite side is obliterated by the iris, which is swollen and pushed forward against the cornea for almost 1 mm. distance. The center of the anterior chamber is obliterated. The iris and pupillary membrane come into direct contact with the posterior surface of the cornea; in this portion, the only remnant of the chamber is a shallow, spindle shaped space on either side of the anterior synechia.

On the injured side of the eye, the base of the iris and the corona ciliaris have been ruptured, and the space is filled in with inflammatory cells, which consists chiefly of lymphocytes, plasma cells, fibroblasts and endothelial leucocytes. Degenerated pigmented epithelial cells are seen scattered thruout the inflammatory exudate. The iris structure and processes of the ciliary body have entirely disappeared in this area. The remaining iris on this side corresponds to the central and pupillary portion. In this portion, the anterior surface of the iris is covered by a layer of fibroblasts and new formed fibrous tissue. The anterior border layer is markedly thickened by edema and a moderate infiltration of lymphocytes and plasma cells. The serum and cells have widely separated the supporting fibers and chromatophores. In several places, there are collections of lymphocytes, and polymorphonuclear leucocytes around the blood vessels. Many of the polymorphonuclear leuco-

cytes have pigment granules in their protoplasm. The pigmented epithelium of this portion of the iris has undergone disintegration, and the granules have been set free. The sphincter muscle fibers show cloudy swelling. The pupil is entirely obliterated by an organized pupillary membrane. The iris on the uninjured side of the eye is swollen by edema. There are several nodules of lymphocytic infiltration around the blood vessels. The pigment epithelium has degenerated in some areas and in others is separated from the iris by edema. (Socalled cysts of epithelial layer.) The pigment epithelium of the iris is separated from the lens capsule by a distinct layer of new formed fibrous tissue, which extends backward to the equator of the lens in the region of the ciliary processes. The posterior chamber, on the uninjured side, is entirely filled in with a partially organized exudate, which contains lymphocytes, disorganized pigment cells, and a few polys. The muscular portion of the body is swollen by edema, and in what formerly was the vascular layer, there are nodular masses of lymphocytes with a few endothelial cells in the center of the nodule. The pigment epithelium has entirely disintegrated, and only a wavy mass of disorganized pigment remains. An organized cyclitic membrane surrounds the equatorial portion of the lens. The lens capsule has been ruptured on the injured side of the eye, and the whole equatorial portion of the capsule is absent in this location. The anterior portion of the lens capsule is present; its epithelium has disappeared, and a thick layer of new formed fibrous tissue lines its inner surface. Posterior to this, a few cataractous lens fibers are still adherent to the fibrous tissue. The capsule has retracted posteriorly into numerous folds. On its inner surface, some new formed fibrous tissue and cataractous lens fibers are still adherent. The remainder of the lens substance was lost during cutting and imbedding.

The radial muscle fiber layer and the vascular portion of the pars plana of the ciliary body are markedly swollen by edema.

The whole choroid is edematous, and several nodular collections of infiltrates are seen scattered thru it. These nodular masses vary in size and are made up of scattered collections of loosely arranged endothelial cells, which are surrounded by lymphocytes and a few polymorphonuclear leucocytes. An occasional giant cell is found in the nodules.

The retina is completely detached from the choroid and shows an extreme degree of cloudy swelling and advanced disintegration.

The remaining vitreous cavity is only a small triangular area, extending from the posterior lens capsule to the anterior surface of the detached retina. It is filled with lymphocytes, a few polymorphonuclear leucocytes, and fibroblasts.

Diagnosis. Trauma of the eye with rupture of the sclerocorneal lamellæ at the limbus, and rupture of the base of the iris and ciliary body on the injured side. Rupture of the lens capsule and traumatic cataract. Edema of the cornea and marginal keratitis. Partial obliteration of the anterior chamber, due to swelling of the injured lens. Nodular cyclitis and choroiditis. Detachment of the retina. Early changes of sympathetic ophthalmia.

The above report is a good clinical and pathologic picture of a moderately severe case of sympathetic iridocyclitis. To this might be added that of a second case following a cataract extraction, in which the sympathogenic eye proved eventually to be the better eye. Also a possible third case, or at least a very suggestive case, following a Mules' operation, in which the fellow eye suddenly developed a dimness of vision, slight ciliary injection and a definite blurring of the edges of the optic nerve, all of which promptly cleared up following the removal of the scleral cup and gold ball. These are the only two cases, with the doubtful third, which the author has ever experienced in his own practice, in observing many hundreds of perforating injuries of the eye sustained in the mining and steel industry, and those following operations. To the above,

however, may be added the experience of having closely observed three other cases in the practice of a colleague. Two were elderly patients following cataract operations, and a third, a boy with a perforating wound: in all of these, both eyes were lost.

This experience reflects no doubt, that of most ophthalmic surgeons. One author has stated that an ophthalmic surgeon of average private clientele would probably see five or six cases in an entire life practice. If this be true, and it likely is, sympathetic iridocyclitis, if not rare, certainly is quite unusual. As a matter of fact, it seems likely that the percentage of cases is lessening, either definitely owing to better care, or relatively owing to better classification. The frequency of such cases, relative to the large number of injuries in which the eye capsule is opened, certainly adds a mystifying element to the problem, in view of the fact that undoubtedly many of such injuries are septic. This point, together with the remote interval which sometimes obtains, and also the fact of the occurrence of sympathetic disease sometime after the removal of the injured eye, together with reported cases in which no discoverable wound was present, such as Meller's three cases in which sarcoma of the choroid was observed, leads one to seriously question whether the disturbing agent really always finds entrance at the point of injury. In contemplating these questions, it causes one to speculate as to the possibility of a certain number of reported cases being iridocyclitis coincident with an injury and not necessarily related to, or dependent upon it.

Most text-books state that a prerequisite is that the capsule of the eyeball of the sympathogenic eye must have been opened, either by trauma, ulcer, or following cataract operation. Yet, Meller and others have reported authentic cases, studied histologically, in which the eyeball had not been opened. In Meller's case the eyeball contained a sarcoma. In Bohm's case there was traumatic dislocation of the lens, in which there was no opening of the eye capsule. It seems to the writer, that in these advanced times one should not adhere too strongly to the name sympathetic ophthalmia. The name has been handed down largely owing to our ignorance regarding the specific underlying causative factors.

Schirmer, Rugge and Uhr are credited with first describing the typical histologic findings in sympathetic disease. Fuchs has contributed a large literature on this branch of the subject. In 35 cases examined by Fuchs, in which there were implications of the second eye, the lesions were considered to be typical. These, briefly, are infiltration with mononuclear round cells, epithelioid cells and giant cells. The round cells were probably leucocytes, the epithelioid cells were derived from the normal cells of the uvea, or from the blood vessels, or from the ordinary connective tissue cells, and also from the pigment cells. The specific infiltration in the sympathogenic eye is identical with the one in the eye secondarily affected. The absolute identity of the anatomic picture in the sympathogenic and sympathizing eyes can be explained only on the theory that the same specific agent attacks both eyes. Fuchs believes that the pathologic agent reaches the second eye thru the circulation, because the optic nerves and ciliary nerves show no signs of transmission, while the manner in which the leucocytes invade the walls of the vessels of the ciliary body and choroid of the sympathogenic eye indicates the probability of a metastasis, exactly as this occurs in tumors.

This specificity of the histologic picture of sympathetic ophthalmia, as described by Schirmer and Rugge and later by Fuchs, has been questioned, based upon the absence of these findings in many cases of clinically typical sympathetic ophthalmia. In a series of 200 eyes enucleated for fear of sympathetic inflammation, Fuchs found that only 15% showed the typical findings. Another confusing feature as to the specificity of histologic findings is Botteri's report of a case of idiopathic iridochoroiditis, in which the anatomic examination showed changes in

the uveal tract exactly similar to those described by Fuchs as typical. He also describes in more detail a similar case, which Fuchs has briefly referred to in his original article. He considers the cases give further proof of the fact that a chronic iridocyclitis may show the same histologic picture as sympathetic inflammation, where there has been no perforating injury or involvement of the fellow eye.

Meller endeavors to reconcile these two diametrically opposed opinions by detailing the clinical histories and pathologic findings of seven cases of this type. Very ingenious explanations are offered but nothing convincing, altho he stoutly defends the specificity of the process. Careful study of two of these cases of clinically typical sympathetic ophthalmia, with absolute lack of characteristic histologic findings, led him to the conclusion that a transference of the disease from the sympathogenic to the sympathizing eye may occur before the disease is developed, in a specific sense, in the sympathogenic eye. In other words, the disease is specific only after it has completely developed, while in the initial stages, it may resemble any other chronic uveitis.

There are two principal theories in explanation of the transference of the inflammation from the sympathogenic eye to the sympathizing eye—the nonbacterial and bacterial. The nonbacterial is the cilio-neural theory of Schmidt-Rimpler; irritation of the ciliary nerves causing reflex disturbance of the circulation, thus interfering with proper nutrition of the fellow eye. The optic nerve theory assumes that the inflammatory process is transmitted along the nerve, crossing the chiasm to the fellow eye. These theories do not seem to have gathered a very large following. Of vastly more importance is the bacterial theory. The clinical history, course and pathologic findings all point more strongly toward a solution of the problem by assuming that it is a germ disease. With regard to the path followed or origin of the agent and its exact nature, there is considerable discussion. The mass of literature on the subject is testimony on this point.

Leber and Deutschmann proposed the migratory theory; that is, the passage of the bacteria or agent along the nerve sheath by way of the adjacent lymphatics. To this there are certain unexplained difficulties as, for example, the disease is occasionally observed following removal of the eye or section of the optic nerve; and this at considerable interval in certain instances. Then too, if it followed this course it is difficult to explain the freedom from meningitis.

A more logical bacterial theory, which seems to hold the greatest encouragement for a solution of the problem, is one which comprehends the possibility of an infective agent being recruited from a source either in, or other than the original injury, the agent being conveyed by the general blood and lymph channels. This allows of the possibility of an explanation of the long interval between injury and onset in the sympathizing eye, particularly in cases where the sympathogenic eye has been removed before the onset of the secondary inflammation. It also admits of the possibility of the agent being of endogenous or ectogenous origin.

The metastatic theory proposed by Berlin has been further investigated by Roemer. From the latter's extended study of the subject, he concludes that the only conceivable paths of propagation from one eye to the other, are the lymph channels of the optic nerves and thru the blood of the general circulation. What he regards as the characteristics of the exciting agent is its ability to remain potent in the eye, or body, for a long period, and an inability to harm other organs. There are only two hypotheses that can be drawn from clinical experience concerning its nature; (pathogenic agent) it must be one of the morbific agents that retain their vitality for a long time in the eye and in the organism, for otherwise the long duration of the sympathetic inflammation, its tendency to recurrence, and the fact that it may appear many weeks after

the commencement of the disease in the sympathogenic eye, cannot be explained. It must also be a germ that is not pathogenic to the same degree to other organs of the body as to the eye; indeed it is highly probable that it is not infectious to any other part of the body.

Regarding the type of germ, Deutschmann has suggested a staphylococcic strain owing to its more frequent association with subacute or chronic inflammatory processes. In view of the wide distribution of tubercular infection and its tendency to produce subacute or chronic processes, it is not beyond possibility of its being the offending agent, even if the bacillus has never been recovered from the lesion. To this possibility Meller lends some encouragement in his report of the clinical histories and histologic findings in the eyes of two cases of sympathetic ophthalmia, which showed necrosis in the sympathogenic eyes, the occurrence of which has so far been emphatically denied. In both, iris and ciliary body were supplanted by a tumor of granulation tissue, consisting of nodules of epithelioid cells and numerous giant cells, surrounded by a wall of lymphocytes. The interior of many nodules was filled with detritus, minute granules and lumps of pigment. This necrosis was due to the complete obliteration of the vessels. The tumor extended to the ora serrata, crowding the retina toward the interior. In the parenchyma of the choroid were also nodules of epithelioid cells surrounded by densely accumulated lymphocytes, which farther backward caused a more uniform thickening. The optic disc was very much swollen, a neuritic ridge displacing the retina. The central vessels were surrounded by small celled infiltration, and on the inner surface of the disc was a fibrinous exudation.

Thru the discovery of necrosis in these cases, one of the most important points of discrimination between tuberculosis and the products of sympathetic inflammation has been removed, and the question revived of relation of sympathetic inflammation of the sympathogenic eye to tuberculosis. This is discussed in detail, and answered in the negative by comparing the remaining histologic and clinical data. Tubercle bacilli were never found in the most exuberant uveal exudations, and the numerous attempts of implantations into animals receptive for tuberculosis were not successful. Both diseases have in common that sympathetic ophthalmia has a predilection for children as well as tuberculosis of the iris, which is most frequent up to the sixth year. Either disease has also been observed following injury. Meller states that all this is not sufficient to render a connection of both diseases probable, but that it may permit claiming a bacterial origin of the inflammation in the sympathogenic eye.

From a study of the literature devoted to the subject of sympathetic disease, there are prominent features which not only lend color, but are very convincing that the infection is frequently endogenous. In only a small number of eyes removed for fear of sympathetic disease does a histologic study of these eyes show characteristic anatomic changes. Many of these cases are not followed by inflammatory changes in the fellow eye for years afterward. If we assume the infection entered at the point of injury in these eyes, the process of the exciting inflammation must have been present as a chronic inflammation and should be capable of anatomic demonstration. This, however, is not the case, for frequently at the time of enucleation there is no evidence of a specific inflammation. In injured eyes showing typical inflammation, the transference which usually always occurs takes place in a comparatively short time. Following an injury the eye may develop a soil possessing the proper conditions for an exciting inflammation, which may actually be produced in an endogenous way months or years subsequent. The organism developing in the uvea of the first eye becomes so highly pathogenic that by metastasis the uvea of the sound eye is affected. A further fact in favor of endogenous

inflammation of the sympathizing eye is its occurrence in cases of shrunken or destroyed globes, in which the first diseased eye is clinically absolutely free from irritation and yet the fellow eye develops the disease.

Of other theories which have been suggested, there is the toxin theory of Eversbusch, in which toxic products are transmitted to the second eye, either directly, or by injury of the vasomotor ganglia or by reflex action. The cytotoxic theory is based on the theory of the splitting of the proteins in the uveal tract. This was advanced by Brown Pusey. Perhaps the most promising theory of the biochemic class is anaphylaxis, as put forward by Elschnig and contributed to by Kummell and A. C. Woods. It is a theory relating to the disintegration of inflammatory tissue leading to absorption of uveal tissue, causing a hypersensitiveness of the uvea of the sympathizing eye.

At the recent meeting of the American Ophthalmological Society, Knapp and Woods discussed this phase of our subject. They injected seventeen cases of uveal tract injury with uveal pigment obtained from the immune reaction which followed intraocular injuries. Eleven showed positive complement fixation reaction. These cases healed normally. Four showed negative reactions, one showed clinical malignant sympathetic disease, three sympathetic irritation. The remaining two showed negative reactions. These injured eyes were removed as a precautionary measure. Three cases of old sympathetic disease showed negative reactions. This, however, does not seem to account for the occurrence of the disease years afterward. Neither does it seem to explain why in certain instances the disease is more virulent in the sympathizing eye than the eye originally involved.

Clinical and laboratory observations, as we see, have evolved many theories regarding the occurrence and transference of the disease from one eye to the other. It is more than probable that there are elements of truth in each theory, which, if we were able to fit them together, might offer a solution. The most promising seems to the writer to be the germ theory, either endogenous or ectogenous, with the circulatory system as the path of transference. While it is admitted that certain diseases have a specific cause, yet there are many affections which may have a variety of causes, or at least the source of the infection, or agent, may come from one of several foci. The same focus of infection may be manifested in a variety of ways, in different tissues. In any event, there are few which show onset, development and effect precisely the same. They are modified by many conditions dependent upon individual metabolic characteristics. Having these facts in mind, are we justified, in view of present knowledge, in believing that sympathetic iridocyclitis is an entity, due to one specific cause finding entrance solely at the point of injury? Rather should it not be considered more as a secondary disturbance, due to a local changed metabolism, the source or the agent being in the uveal tract of the sympathogenic eye, or perchance, in some other part of the body.

Possibly there may be a local focus of infection at the point of injury, but can it be successfully stated that this is always the case, and that this focus is always the cause of inflammation in the fellow eye? Injury of a tissue, particularly if there follows a low grade inflammatory process, leaves that tissue more susceptible to bacterial invasion or toxic agents, the resistance having been lowered. It is conceivable that bacteria may enter the globe at the time of injury, remaining resident and more or less quiescent for an indefinite period. This is likely true in a certain number of cases, but it is equally conceivable that injury to the uveal tissue prepares a fertile soil for bacterial invasion from other foci of infection. The resulting products of this invasion finds elements in the fellow eye for which it possesses a definite affinity, the severity of the attack being in accord with its virulence. This

seems true particularly in instances where long intervals are recorded between the injury and onset of sympathetic iridocyclitis.

The fact that in certain cases even following enucleation, at more or less remote times, sympathetic iridocyclitis is established, seems to indicate, at least, that while we may grant the influence of the injured eye in possibly establishing a low grade inflammatory process, or possibly preparing the soil in the fellow eye, the continuance and increasing severity some time after enucleation are more than significant that the "fire" is being fed from another source. This may be from a small apical tooth abscess containing perhaps a strain of staphyloccoci, with special affinity for uveal tissue, rendered so by the influence of the original injured uveal tissue. The numerous relapses which sometimes occur during the course of the disease may be accounted for in the same way.

In this connection it is well to consider the possibility that, even if an eye has undergone an attack of sympathetic iridocyclitis with recovery, subsequent inflammatory relapses may be the result of entirely different causes, and in no way dependent upon the original inflammation, except in so far that an uveal inflammation renders the eye more subject to subsequent inflammation, the exciting cause being in no way related to the original cause.

Crediting enucleation, in a given case, as the means of having saved the fellow eye, seems to the writer difficult to prove. We remove an eye, believing it to be a menace, yet, in anticipating danger we have no clinical or laboratory means of definitely knowing. If the inflammatory changes have once become established in the fellow eye, and the inflammatory process in the sympathogenic eye not so severe as to anticipate complete destruction, and the sight previously servicable and not greatly reduced, such an eye should not be enucleated. It may prove to be the more serviceable.

It is difficult to believe that the causative agent of sympathetic iridocyclitis is always resident in the sympathogenic eye alone, and that it is the sole etiologic factor to be considered in accounting for the changes in its fellow. If it were, how can those cases be explained in which useful vision is retained in the sympathogenic eye, and yet the sympathizing eye is lost? It seems reasonable to believe, that, if the agent is virulent enough to destroy the sympathizing eye, surely it would produce a like result in the sympathogenic eye, especially in view of the fact that its uveal tissue has already been damaged. Yet, this very fact leads us to consider a possible explanation in assuming that it is an anaphylactic reaction contributed to by infection from various focal points. At the time of the injury, the injured uveal tract gradually develops a degree of immunity, thus lessening the severity of the attack in the sympathogenic eye, while in the sympathizing eye invasion takes place before the eye has had an opportunity to establish any degree of immunity, thus permitting a much more severe attack.

The case history cited at the beginning of this contribution seems to point strongly to the belief that the real infection emanated from the unhygienic oral state, the injury acting as the determining factor in producing a soil suitable for the propagation and development of bacteria having an affinity for uveal tissue, the pathogenicity increasing until the noxa attacks the fellow eye. In the matter of therapeutic measures instituted, certainly not any were consistently carried out, save that of thoroly cleansing the oral cavity.

TRACHOMA AND OUR END RESULTS.

JOHN H. BURLESON, M.D., F.A.C.S.

SAN ANTONIO, TEXAS.

The writer believes that trachoma includes many forms of conjunctival inflammation and certain results produced. These results include especially hyalin degeneration and cicatricial deformity of the tarsus. The treatment of acute cases should be soothing. When deformity of the lids has occurred, he prefers the slicing down of the tarsus from the outer surface as recommended by Cary. Read before the Texas State Medical Association, May, 1922.

I am minded of the fact, that to differ with an accepted theory is thought by some to be medical heresy. I am also minded of the fact that medical precedent hunters have not made medical history. The views expressed in this paper open a new line of thought concerning this disease.

The points I wish to stress are, first; that trachoma is not a disease entity. Second; this disease as we see it, is a symptom complex, the result of any bacterial infection of the conjunctiva. Third; it is the duty of ophthalmologists to standardize diagnosis as much as possible for the benefit of the school physician, the public health nurse, and the laity.

The essential element of the cause of trachoma bodies is no nearer a solution than it was before the advent of the microscope. The two views held which are entitled to consideration are: first, that they are new growths of special pathologic character; second, that they are natural lymphatic follicles, and their changes are due to repeated attacks of inflammation.

I have always looked with suspicion upon any diagnosis that was not founded on fact. That the disease is of microbic origin has not been proven. In the absence of this proof I have come to the conclusion, after a long and varied experience with trachoma as we see it, that the changes in the lid are due to repeated attacks of inflammation and not to a specific trachoma infection.

I am of the opinion that the socalled second and third stages of trachoma are not due to a morbific agent, but due to amyloid degeneration from chronic inflammatory changes in the tarsus. The final stage of this disease, cicatrization, is best described by de Schweinitz who says, "By a gradual process of cicatrization of the old granulations, and successive cicatrization of new crops, a chronic induration and diffuse scar tissue results; this being attached to the tarsus, which itself has undergone softening thru lymphoid infiltration, contracts and the deformity of the lid so common in this disease results." It is to this softening and later contracting of the tarsal cartilage that I wish to call your attention.

I believe that any specific infection of the palpebral conjunctiva, if allowed to become chronic, either thru bad treatment or neglect, will produce a low inflammation in the tarsus and produce permanent changes in that structure.

I would call your attention to the structure of cartilage. It has a semiopaque, nonvascular tissue base, the matrix of which contains nucleated cells which lie in cavities or lacunæ of the structure.

Prolonged inflammation of the conjunctiva from any infection, due to continuity of tissue, must inflame the tarsus, producing chondritis, which is followed by a shrinking and distortion of the cartilage. In the early stages of chondritis we have the thickening of the lid and the presence of organized nodules known as trachoma bodies imbedded in the tarsus; this condition is not due to specific infection, but to inflammation.

The different varieties of granulations are described according to the different stages of inflammation and to the clinical picture found at the date of examination. Papillary trachoma, follicular trachoma, mixed trachoma, acute granulations, chronic granulations. Examine your patient and take your choice as to nomenclature. Acute trachoma, viewed from a purely clini-

cal standpoint, I must admit I have never been able to diagnose as such.

The clinical picture of socalled trachoma is identical with any acute granular disease of the conjunctiva from whatever cause. I find the same difficulty of diagnosis is experienced by most men with whom I talk; as one tersely expressed it, "When acute granulations get well, they are not trachomatous, when they don't they are." He should have said when acute granulations get well the tarsus has not become involved, when it has they don't.

The chaotic state in which we find ourselves in diagnosing granulations of the conjunctiva, is a reproach to our specialty. It is incumbent upon us to give the cause of the condition; this can only be done by painstaking care. The microscope should be used much oftener to determine the offending organism; it is not enough to evert the lid and gravely give the diagnosis of granulated lids or trachoma.

Is it any wonder that the school nurse or school physician should be guilty of this, if we ourselves are guilty? We all know the great injustice often done to children from such a diagnosis; in loss of time from school, and financial loss to the parents for useless surgery and treatment for the cure of follicular conjunctivitis.

TREATMENT.

Treatment should be considered from two viewpoints, acute and chronic. In the acute or primary stage, every effort should be made to determine the kind of infection you are dealing with, and appropriate remedies used to stop infection. It is my custom in acute inflammations of the conjunctiva to use strong astringents and mild antiseptics, the removal of all sources of irritation, i. e. to prohibit the use of the eyes for work, this to eliminate eyestrain, the use of colored glasses to prevent irritation from light and dust, the frequent application of cold compresses, and the instillation of a weak solution of atropin to relieve ciliary congestion; in fact to use any remedy which will assist in allaying inflammation and prevent it from becoming chronic. The careful handling in this stage, in my opinion, will often prevent the development of the second and third stages, chondritis.

Under the best of conditions, chondritis is a tedious disease to treat and relapse may be frequent; but greater success will attend this line of treatment than any other I have tried. I am firmly convinced that caustics, such as copper crystal, phenol solutions and strong bichlorid solutions or, 'grattage" whether the instrument used be forceps or roller, should never be used in acute infections of the conjunctiva.

The object of our treatment should be the prevention of scar tissue and not the formation of it. I believe the administration of alteratives and tonics during acute inflammatory conjunctival infection will promote resolution.

If your case is first seen in the degenerative stage, and cicatrization with the formation of scar tissue has already begun, your treatment can only be surgical and it is the end results of surgical measures I wish especially to discuss.

The earlier methods of operating upon trachomatous lids were the use of some form of "grattage," brushing with a stiff brush; or the use of instruments devised to express the granulations. The two most frequently used are the hollow forceps devised by Noyes, and the roller forceps of Knapp. The object of "grattage" was to promote resolution by squeezing out the socalled trachomatous bodies. This line of treatment, will in a certain percentage of cases give excellent results, provided there has been no definite changes in the shape of the tarsal cartilage. The greatest good, however, that results from "grattage," after a chondritis has developed, is that it hastens the stage of cicatrization and prepares the lid for more radical measures, saving the patient much time and suffering.

The object of all radical operative procedures upon trachomatous lids is the removal of the pathology caused by chronic inflammation, and as most

of the change has occurred in the tarsal cartilage, operations upon this structure are most frequent.

Tarsectomy is done by everting the upper lid, placing the distorted and buckled cartilage on the stretch and held in position by means of a spatula. A sharp scalpel is then boldly passed thru the entire thickness of the cartilage along its entire length, it is then severed from its attachments and removed in its entirety. The denuded lid is then covered by drawing down the retrotarsal folds and sewing them to the lid.

The different modifications of tarsectomy are as varied as are the men doing them. I believe, however, that most operators have abandoned the stitching of the conjunctiva for the reason that it occasionally produces a deformity of the lid and is also likely to prevent the correction of the existing entropion. My own experience has been that the cases do better that are not stitched, but in these you may get some conjunctival adhesions to the eyeball.

Several years ago at a meeting of the State Medical Association, Dr. Cary of Dallas read a paper before our section describing an operation he was doing upon the tarsus, which for want of a better term I shall call "tarsotomy" to differentiate it from tarsectomy, which we all know. At the time the doctor read his paper, I must admit I did not get a very clear idea of his technic, but afterward I had the pleasure of seeing him operate, and was so much impressed with its mechanical possibilities, that I have used it to the exclusion of all other operations and my results have been much better.

This operation, unlike tarslectomy, is done upon the outside of the lid. The first step is the application of a large chalazion forceps to protect the eyeball and to control hemorrhage. Thru the fenestrated opening above, the skin is incised the entire length of the lid and parallel to its free margin, this incision exposes the fibers of the orbicularis muscle; the skin and muscle are then freed downward to the margin of the lid and upward to the extreme upper border of the tarsus, in this manner the entire tarsus is exposed. After the muscle is separated, its fibers are grasped with forceps and removed with scissors. In most cases the exposed tarsus will be found to be unusually thick and curved convexly forward.

The tarsus is now removed in thin slices, by the use of a very sharp scalpel held flat against the convex anterior surface of the thickened tarsus. The success of the operation depends upon the thoroness with which this is done. There is little danger of cutting thru, but no harm is done if this accident should occur; the margin of the lid is left intact. After the tarsus is thoroly straightened, the wound is closed with only a skin suture, a light dressing is now applied, to be changed the following day, and the stitches removed on the fourth day.

The advantages of this operation over others are: (1) you do not get a deformity of the lid; (2) conjunctival adhesions are prevented; (3), you have no granulating wound; (4), involvement of the cornea after operation is not nearly so likely; and, (5), relapses are far less frequent. I wish, after a thoro trial, to commend this operation as being a distinct advance in tarsal surgery.

In conclusion I wish to quote an old adage: "That a prophet is never without honor, save in his own country," and venture the opinion, that if this operation had been perfected by some ophthalmologist with an unpronounceable name from Central Europe he would have many advocates among us.

NOTES, CASES AND INSTRUMENTS

CASE OF PULSATING EXOPHTHALMOS.

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The following cured case of pulsating exophthalmos was exhibited at the May meeting of the Oregon Academy of Ophthalmology and Otolaryngology:

Miss E. M., 24, received a blow on the angle of the right lower jaw ten months ago; she lay senseless for a time, and was aware of a loud rushing tinnitus in the right ear on awakening. The only external evidence of injury was a slight skin bruise. The ear noises caused her to visit Dr. C. C. Wilcox, for whom we examined her, a week after the injury.

At this time there was no exophthalmos, but diplopia from right IV. paresis; vision O. U. 20/20. Tinnitus was increasing, of a throbbing character; there was no tympanic or intraocular abnormality. Diagnosis of probable traumatic cavernous-carotid aneurysm (right) was made. The patient left observation for three weeks. At the later date, a month from the injury, exophthalmos and venous congestion had begun in the right eye: V. O. D. 20/30, O. S. 20/30. A distinct bruit was audible over the right zygoma with the stethoscope. Tinnitus had increased to a degree that prevented sleep. There had been 20 pounds loss of weight.

The patient was returned to Dr. Wilcox with the suggestion that the right carotid be tied unless digital compression gave immediate relief. She did not follow orders, but left the city, falling into the hands of a chiropractor, whose "adjustments" were made in spite of increasing exophthalmos, paralysis of the right III., IV., and V., and what is described as corneal anesthesia and bullous keratitis lasting several weeks.

When next seen, five months after the injury, both eyes protruded and pulsated under the finger; V. O. D. 20/70, O. S. 20/30. A caput medusae of huge veins encircled both corneae. The aneurysmal bruit was audible to the unaided ear at four inches from the right zygoma. Media were clear; there was only slight broadening of the retinal veins. Lateral excursion of both eyes was much limited, and diplopia was constant. There had been a total loss in weight of fifty pounds. Immediate operation was again recommended. The patient did not, however, return to the care of Dr. Wilcox for three months more, eight months after the injury, in a most hopeless and unhappy mental state.

Ligation of the right internal carotid—a most difficult procedure—was performed a few days later, using four ligatures, applied so as to cause slowing of the current and thrombotic occlusion over a considerable length of vessel wall.

The patient awoke to find her head noises completely gone; diplopia disappeared in four days, a slight mydriasis persisting for several weeks. Exophthalmos and venous dilatation have gone down to less than a third of that seen prior to the ligation. No bruit is heard over the zygoma. Vision in ten days was 20/30 O. D., 20/40 O. S.; in a month 20/20-2 O. D., 20/30 O. S. There is some hyperopia and convergence insufficiency, corrected by glasses. The gain in weight of fifteen pounds reflects a complete change in mental attitude and justifies the wisdom of Dr. Wilcox in tying off the internal carotid rather than the easier common trunk, with its possibilities in the way of cerebral ischemia.

CHANGES IN REFRACTION.

HARRY FRIEDENWALD.

BALTIMORE.

Marked and rapid changes of refraction are observed in a variety of ocular diseases (developing cataract, glaucoma, cyclitis, etc.), and as a consequence of too sudden withdrawal of food in diabetes. But the case which the writer reports is unique in his experience and he has been unable to find

any reference to a similar one in medical literature.

Mr. I. H., aged 43, was first seen July 18, 1913, complaining of some difficulty in near vision. His eyegrounds were normal and his distant vision perfect, 20/12+ in each eye (some of 20/9 and 20/7.5). He accepts +0.25 D. C. in the right eye, but no plus glass in the left. R. E. +0.5, L. E. +0.25 were ordered for reading.

He returned Sept. 28, 1914; condition unchanged except that neither eye accepted plus glasses, and that +0.75 was required for each eye for reading.

In Sept., 1915, and in Jan., 1917, he returned, the examinations of the eyegrounds showing normal conditions and central vision unchanged. +1.25 D. S. was ordered in 1915, +1.5 in 1917.

December, 1919, the same conditions were found, and +2. D. S. was found necessary for reading.

The patient returned July 8, 1921, complaining of pain in the right eye during the past two days, with some discomfort in the left; sudden movements are painful. There was a small patch of slight episcleritis in the right eye. The pupils were equal in size, the eyegrounds normal. Hot applications were applied. The affection was so mild that no other treatment was deemed necessary.

On July 11, the congestion had disappeared, there was still some edema of the conjunctiva and at time some pain.

July 18. All signs of inflammation have disappeared, but there is still pain in and around the right eye. Eyegrounds and tension normal. An examination of the central vision showed R. E. 20/96, 20/76 doubtful, +2.= 20/12, L. E. 20/12 accepts +0.5. These glasses were ordered with the object of relieving the discomfort of which the patient was complaining, and +2. additional were given for reading.

The patient returned on July 22. With the correcting glasses the vision of the right eye had fallen to 20/24 and +2.5 was required to bring vision up to normal. This glass was ordered. On account of the rapid changes in the refraction the urine was examined and found normal.

July 30. The hyperopia of the right eye had again become reduced to +2.

Aug. 21, the patient returned stating that the condition had changed again and V. was found R. E. 20/19 +0.75 20/12, L. E. 20/12 +0.5 accepted. The patient feels now quite comfortable without glasses. With +2.5 each eye reads finest print with equal ease.

Inquiry now showed that the dentist Dr. A. C. Brewer had, by X-ray examination, found an abscess in a lower molar tooth. This was extracted on Aug. 7. The patient was convinced that the improvement in the condition of his eyes followed directly after the removal of the tooth, but it must be noted that a week before the extraction there was a slight lessening of the hyperopia.

On May 31, 1922, vision of both eyes was equal (20/12), each eye accepting +0.50; the near points of both eyes were alike. The patient has remained perfectly well.

In this case the affection was monocular. It is to be noted that the rapid increase of hyperopia of about 1.75 D. was observed about a week after the occurrence of what was looked upon as a mild and rapidly disappearing episcleritis. The hyperopia continued to mount and four days later the increase was 2.25 D.; eight days later the hyperopia had again diminished 0.5 D. and after a further period of three weeks it had almost returned to the normal condition. During the entire period, the accommodation did not vary in the two eyes.

It may be suggested that there was possibly a cyclitis accompanying the episcleritis, but there was no evidence of this, either in the vitreous or on the posterior surface of the cornea.

The writer does not venture to explain the case. The relation between the eye affection and the tooth may be simply coincidence. Still, in the absence of any satisfactory explanation, the fact dare not be brushed aside without consideration.

CENTRAL SCOTOMA WITH PYORRHEA.

A. G. Hovde, M.D.

SUPERIOR, WISCONSIN.

Mrs. R. E., aged 40, feeble, occupation housewife. Referred January 21, 1921 to have glasses fitted. Has noticed a gradual failing of her sight the last three or four months, and lately she has had some pain in the eyes. Pupils probably slightly dilated (3.5 mm.); react to light and accommodation. Cornea, iris and anterior chamber normal. The lens also appears clear.

At the trial case, R. vision 1/10; L. 3/10. No lenses could be found to improve the vision in either eye.

The media were clear in both eyes. The fundi appeared normal with the exception that the outer upper quadrant of the papillæ seemed rather pale in color.

Peripheral fields normal. Small central scotoma for green and red. The size of the green test object was 2 mm., which in one position was called white. The moment the test object was moved to either side its color was recognized. The red test object in the same position was called brown.

Diagnosis: Toxic Amblyopia.

Further General Examination: I questioned her if she had been taking any medicine, patent or otherwise, and she said no. The same reply she gave to my question if she had been drinking any liquor. My quiz did not reveal any suggestion as to the cause. Examination of throat, nose and accessory nasal sinuses was negative, but she did have a very exaggerated pyorrhea alveolaris. Many of the teeth were practically standing in a pool of pus, surrounded with necrotic tissue. I could think of this only as a remotely possible cause of this patient's toxic amblyopia, and determined to hunt further. I commented upon the poor condition of her teeth and gums, but did not suggest to the patient that this might be the cause.

Since the doctor who referred the patient to me thought that only glasses were necessary to bring back her sight, I sent her back to him for a general physical examination, stating to her that I found some condition of the optic nerve which glasses could not correct, and that we must try to determine its cause.

I called up her doctor and explained to him the situation and urged him to be on the lookout for anything that would lead to a diagnosis. The patient did not return to me until March 10th, when she called, she said, to thank me for my thoughtfulness in pointing out to her what was doing harm to her eyes. She told me that she realized that I was much impressed with the bad condition of her teeth, and she went right to her dentist and had them all removed. Her vision has been improving ever since.

At the trial case, she now reads 8/10 right and left without any faltering. The color of the papillae is not so pale as it appeared previously. There is no central scotoma of any kind.

Discussion. Since no treatment except the dental extraction was administered, we can only suppose that this patient was overwhelmed with the toxin from the bacterial activity and necrotic processes around her teeth.

Hysteria, of course, might have simulated the condition, but she was hardly well enough informed to check up so thoroly with the symptoms of toxic amblyopia. Nor did she look like she was addicted to the use of liquor, and she had no odor of tobacco about her. It is possible, of course, that the patient may have been taking some brand of alcoholic mixture continuously, and from this produce the toxic amblyopia; but even so, complete cessation from its use would hardly bring about so quick a recovery.

SOCIETY PROCEEDINGS

Reports for this department should be sent at the earliest date practicable to Dr. Harry S. Gradle, 22 E. Washington St., Chicago, Illinois. These reports should present briefly the important scientific papers and discussions.

ROYAL SOCIETY OF MEDICINE.

Section on Ophthalmology.

Clinical meeting of Friday, June 9, 1922.

SIR JOHN H. PARSONS, F.R.S., Chairman.

Mass Obscuring Optic Disc.

MR. J. F. CUNNINGHAM showed the patient. Even under the best conditions, the mass could be seen only indefinitely; it involved the disc in its lower part. He thought it was possibly of congenital origin, tho it might have followed upon some trauma.

Discussion. — SIR JOHN PARSONS thought it more likely that it was a congenital film, due to an excess of fibrous tissue on the disc, than that it was ordinary retinitis proliferans.

Symmetric Swelling of Lids.

MR. DOYNE exhibited a case. As the patient was an inmate of a mental hospital, there might be doubt about the history he gave. There were symmetric swellings toward the outer margin of the upper lid, in both eyes. The patient himself said they had been there ever since birth, and that they had undergone no appreciable change in size. At first he regarded it as a dermoid, but felt sceptical about that later, because on pressure it gave a sensation of lobulation, such as from a lipoma. He asked for opinions.

Discussion. — MR. J. B. LAWFORD thought cystic swellings could be excluded. Under pressure the tumors receded very readily, so that it was difficult to make out their outlines with the finger tips. And against the idea of dermoid was the fact of the swelling having been stationary in size for forty years; he believed orbital or extra-orbital dermoids were never stationary, some grew quite rapidly. In his view, the diagnosis lay between symmetric fatty swellings and a soft form of congenital hydroma. Their extreme softness favored the former view.

MR. MALCOLM HEPBURN thought the possibility of it being lacrimal gland should be considered.

SIR JOHN PARSONS said the swellings apparently were similar to those frequently seen in the inner part of the upper lids in elderly people, which were generally recognized as being due to orbital fat coming thru the opening in the facial septum. He was unfamiliar with such swellings in the outer part of the upper lids.

Models for Prothesis.

MR. OLIVER showed some beautifully executed models for prothesis.

SIR JOHN PARSONS said these models showed that the work had been brought to the pitch of a very fine art.

Detachment of Retina.

MR. H. R. JEREMY exhibited a woman, aged 38, who had a retinal detachment at the macula. A fortnight ago, she attended hospital and stated that she had been unable to see with the left eye for two years. When eight years of age, she was struck in that eye with a stone. At times the eye was painful. A fortnight ago she could perceive fingers at the periphery of the field, and now there was only perception of shadows there. The tension of the eye was not raised, there was no conjunctivitis or circumcorneal congestion, and the cornea was clear. There were, however, a few fine vitreous opacities. The disc was normal. There was a detachment of the retina at the macula, ovoid and regular in shape, greyish-white in color, and the surface was smooth. A fortnight ago the detachment was 4 or 5 discs in diameter, the edges were regular, and there was choroidal pigmentation below. The tension was raised, plus 8 to plus 10. Wassermann reaction was negative. He had advised having the eye removed, but thought he would like opinions of members first.

Discussion.—SIR JOHN PARSONS said that if the detachment started in the macula it was a long history (two years).

He referred to a case which was under the care of the late Mr. Marcus Gunn, in which a tiny tumor in the macular region manifested itself at an early stage in the history by a disturbance of the central vision. Examination showed, in the lower part of the fundus, a large simple detachment of the retina, separated from a very shallow detachment just over the growth.

During the meeting, congratulations were offered to Sir Richard Cruise on the honor just conferred on him by his Majesty in creating him a Knight of the Royal Victorian Order.

H. DICKINSON.

COLORADO OPHTHALMOLOGICAL SOCIETY.

April 15, 1922.

DR. C. O. EIGLER presiding.

Multiple Ocular Diseases in Same Patient.

F. R. SPENCER and C. L. LARUE, Boulder, presented a man, aged seventy-two years, who had a variety of ocular disturbances. He had come on April 6, 1922, complaining of a dull pain in the left eye and of blurring of near and distant vision. He said the vision had failed rather suddenly in the past few weeks. The vision was R. 6/20, L. 6/60. There was a large pterygium on each eye. The eyes were noticeably convergent. There was some cortical opacity and a slight general haziness of each lens. Each vitreous contained a few large floaters. The periphery of each fundus showed several fair sized areas of chorioretinitis. Tested with the colored lens there was constant diplopia. Correction did not markedly improve vision. After transplantation of the pterygia, correction gave vision of R. 6/7, L. 6/12.

Discussion. EDWARD JACKSON, Denver. The choroidal change is old and there is comparatively little atrophy for the amount of pigment deposit. The old choroiditis probably had little to do with the recent change in vision.

Coloboma of Iris and Choroid; Retinal Detachment.

W. T. BRINTON, Denver, (by invitation) presented a young man, aged eighteen years, whose right eye had a congenital coloboma of the iris. The vision of that eye had always been poor. Two weeks previously he had suddenly noticed that the vision of this eye became blurred, and that he was unable to see objects to the left of the middle line. On examination the vision of the affected eye had been 16/200, that of the left eye 16/16. The coloboma was found also to involve the choroid, and there was a temporal detachment of the retina extending backward from the periphery, obscuring the disc, and involving about a half of the fundus. There was heterochromia iridis. Contrary to advice the patient had recently engaged in a boxing match, and he had suffered a hemorrhage into the vitreous which reduced the vision of the right eye to light perception, and left numerous floating opacities in the vitreous. A Wassermann test was negative, as was examination of the teeth and sinuses.

Discussion. EDWARD JACKSON, Denver. I have never before seen detachment of the retina in a case of choroidal coloboma. This does not look like an injured eye, and I should rather think of a new growth. The retina seems to be adherent over the coloboma. The detachment apparently covers the optic disc. The detachment may have come on very gradually and may have been unnoticed until it involved the macula.

G. L. STRADER, Cheyenne, Wyoming. Dr. Gifford feels that all detachments due to new growths are the result of injury, altho in many cases they may come on gradually and date back a good while, even occasionally to an injury several years previous.

C. L. LARUE, Boulder, referred to the case of a man who, while in the S. A. T. C., four years previously, had received a blow on the eye from an apple, and who said that the vision of this eye had not fallen for a long time after the blow was received, but who had recently come in with complete detachment of the retina.

Lethargic Encephalitis.

W. H. CRISP, Denver, reported two cases of abducens paralysis, one definitely symptomatic of lethargic encephalitis, the other of uncertain causa-

tion. The first case was in a young woman aged nineteen years. She had complained of headache for about two weeks, but the only other pronounced symptoms were dilatation of the right pupil, and diplopia associated with convergence of the right eye. The vision was unaffected, the right pupil reacted very slightly to light, the accommodation of this eye being, however, unaffected, and the movement of the right eye outward was retarded before the median line was reached, and stopped entirely at the median line. The patient had not been markedly lethargic, but answered questions very briefly. Drowsiness was not pronounced. For a time doubt was thrown upon a diagnosis of lethargic encephalitis because of the extremely frank and almost steady character of the ocular disturbances. But every other line of investigation, including a Wassermann test, proved negative, and the latter course of the disease was slightly more characteristic of lethargic encephalitis, altho practically no other local symptoms developed. There was very doubtful elevation of the right optic disc, but the patient was rather highly hyperopic.

The second case was in a man, aged twenty-one years, who stated that he had been seeing double for about six weeks. This ocular disturbance had begun about the time he got up from a febrile attack, during which the temperature had been for part of the time 103 degrees, and which was accompanied by a marked desquammative eruption on the legs below the knees and on the wrists and hands. The eruptive condition had since been readily diagnosed by two Denver dermatologists as erythema multiforme. There was a doubtful clinical history of syphilis, but repeated Wassermann tests, including one made since the patient came to Dr. Crisp, had been negative. The patient saw double only when he looked to the left, and in direct gaze the convergence measured only 14 centrads; there being apparently a rather moderate weakness of the left abducens muscle. In spite of the negative Wassermann, mercury inunctions and potassium iodid had been ordered. A week later the muscular weakness had improved more than 50 per cent, but the patient announced that for financial reasons he had not used any medicine. Since erythema multiforme was always symptomatic of a severe underlying condition, the question arose what the underlying condition might have been in this case, and what was its connection with the ocular paralysis.

Discussion. EDWARD JACKSON, Denver. There is a possibility that this second patient had an unusual form of lethargic encephalitis. To-day I saw a case in which the disturbance began fifteen months ago. I saw the man about a year ago, when he had complete abducens paralysis of both eyes. He went on for three or four months without any improvement. In the latter part of the summer, I noticed a distinct outward movement in one eye, and now he has outward movement of 15 or 20 degrees in this eye. A week ago I found distinct outward movement of the other eye, and to-day I found the movement in the second eye distinctly increased. He has been going thru some exercises. I do not expect him to recover entirely, and he may need an operation, but as long as he is improving I shall not advise this. I believe the case was one of lethargic encephalitis.

W. H. CRISP said that he had considered the possibility that his second patient had suffered from lethargic encephalitis of exceptional form.

W. F. MATSON, Denver, wondered whether the case might have been one of scarlet fever in spite of the lack of eruption on the body and face.

E. E. MCKEOWN, Denver. I recently had a case of mastoid operation on a girl of six years, in whom it was necessary to tie off the jugular vein. She slept for five days after the operation on the left ear, and the right eye became affected. She was completely unable to abduct this eye. Four days later a similar disturbance developed in the left eye. The right eye began to return to normal after three weeks, but the left eye is still slightly paralyzed. The Wassermann test and all other tests and studies were negative. There was no evidence of facial paralysis on either side.

WM. H. CRISP, Secretary.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

DR. HILLIARD WOOD, President.

May 15, 1922.

Laceration of Nasal Duct.

DR. M. C. CULLOM reported the case of L. R., white, female, aet. 26, who on May 8, 1922 presented herself at his office with an injury to the left lower lid, due to accidental blow from the heel of a shoe at inner canthus of left eye. Examination showed a tear of the

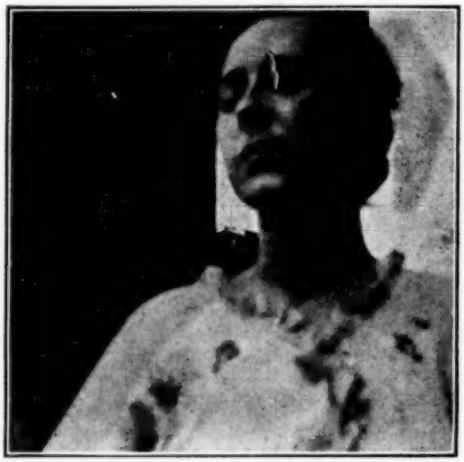

Fig. 1.—Laceration of nasal duct with probe placed in it. (Cullom's case).

lower lid extending along the fornix internally, and along the infraorbital margin externally for about one inch; the tear was completely thru skin, muscle and mucous membrane; the tear duct was torn thru completely.

Patient was removed to the hospital and repair of injury undertaken, the object being to preserve, if possible, a patulous tear duct. In pursuance of this, Bowman's lacrimal probe No. 1 was passed thru the inferior canaliculus, and then thru the tear into the lacrimal sac, and then down thru the lacrimal canal. This approximated the torn surfaces perfectly. The edges were sutured with interrupted silk sutures and the eye dressed with probe in place. The probe was allowed to remain in place one week, at the end of which time it was removed, along with the sutures. The cosmetic effect was good, and the patient seems to have a perfectly functioning duct.

Pulsating Exophthalmos—Report of a Case.

DR. J. LESLIE BRYAN reported the case of R. S., male, aet. 5, referred to him February, 1921, to ascertain the cause of distended veins in the eyelids. History: No serious illness; no injury; patient eats and sleeps well; plays out-of-doors continually. Examination: Well nourished, well formed boy, except a peculiar shaped head. X-ray of frontal sinuses, antra, ethmoids, sphenoids and mastoids negative. Urinalysis and Wassermann negative. Nose negative. Tonsils hypertrophied and diseased. Ears normal. Vision each eye 15/15. Eyes slightly bulging; veins of upper and lower lids of both eyes and temporal regions distended; no pulsations; no bruit at this time, but bruit appeared in a few weeks, and was marked, being heard over any part of the head without use of stethoscope. Conjunctiva, sclera and cornea clear; iris normal; reactions normal; lens and media clear. Ophthalmoscopic examination showed marked neuroretinitis, a typical choked disc, such as is often found in cases of brain tumor; this was binocular. No hemorrhages, no pain, no interference with motility of ocular muscles. Fields normal.

A positive diagnosis was not made until the case was seen for a second time. The doctors all agreed that the administration of increasing doses of potassium iodid was the proper treatment. This was carried out over a period of time without beneficial results. Later a ligation of the left common carotid was done, without relief. Last fall a resection of the superior ophthalmic vein was done on the left eye, a Killian incision was made, the resection being made thru the upper and inner angles of the orbit. This had no beneficial effect on the condition, the result being that the eye was displaced outward and downward, the distension of veins and of the lids was increased, and the veins of the conjunctiva were engorged. When last seen the optic nerves gave the appearance of

beginning atrophy. The father was advised to consult Dr. de Schweinitz.

Dr. Bryan said, that in searching the literature at his command he had found very little with reference to this condition except in the text books, the most concise and comprehensible description being found in the 9th edition of de Schweinitz.

Discussion. Dr. Hilliard Wood said that when he saw the patient May 23, 1921, there was a distinct bruit heard all over the head, most distinct in front. Bruit was not abolished by pressing on either common carotid, but was abolished by pressing on both common carotids at the same time.

Paralysis of Divergence.

Dr. Robert J. Warner reported two cases as follows:

Case 1. J. M., aet. 56, male, complained of double vision past week. Examination shows vision of each eye 20/40; with +1.50 D. S., V.= 20/20; +4.00 for reading. Cornea, sclera, pupillary reaction and fundus each eye negative. Homonymous diplopia and convergent strabismus at 20 feet, single vision with a 20 degree prism, base out; without prism the diplopia decreased as object approached till single vision at 2 feet. Diplopia same in all fields. Blood Wassermann 4 plus. Patient put on salvarsan and mixed treatment. Eye examination three weeks later showed no change.

Case 2. M. P., aet. 38, female. Complaint of double vision for past three days. Examination showed vision each eye 20/15. Cornea, sclera, pupillary reaction and fundus each eye negative. Homonymous diplopia and convergent strabismus at 20 feet; single vision with a 20° prism, base out. Without the prism, the diplopia decreases as the object approaches the eye till single vision at one foot. Diplopia same in all fields. Urinalysis and blood Wassermann negative. Spinal Wassermann requested. Diagnosis of sleeping sickness made by Dr. Witt.

Paralysis of divergence is a rare condition. It is characterized by convergent squint and homonymous diplopia, decreasing as an object approaches the patient, till a certain point is reached, where there may be single vision, while within this limit there may be exophoria. The squint and diplopia remain the same as the eyes are carried to the right or to the left, and this point alone differentiates this condition from a paralysis of abducens, in which the diplopia increases on looking to the right or to the left.

The condition usually develops suddenly, occurs in all ages, and more often in hyperopes than myopes. As to cause, cerebral hemorrhage, nephritis, with high tension, tabes dorsalis, and multiple sclerosis are given. The exact location of the divergent center is unknown, but it is thought to be at the base of the sixth nuclei, at the floor of the fourth ventricle, near the nuclei of the seventh nerve.

Discussion. Dr. Fred E. Hasty said that in the majority of cases of sleeping sickness he had observed, the eye complications are usually first those of the muscles supplied by the third nerve.

Dr. E. B. Cayce reported two cases of sleeping sickness in which muscle disturbances were the first symptoms to be observed. In both cases the muscle trouble subsided after the disease had run its course.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

June 13, 1922.

Foreign Body in the Orbit.

Dr. A. C. Lewis reported the case of Mr. J. B., who, on April 2nd, 1922, removed the cap from a 38 pistol cartridge, laid it on a stone and struck it with an iron rod. It exploded and struck him in right eye, cut thru upper lid at the margin and removed a large semicircular piece of it. The cap penetrated the bulbar conjunctiva and Tenon's capsule about 1 cm. from the nasal limbus, and just above the tendon of the internal rectus muscle. The X-ray showed it well back in the orbit lying over the ethmoid region. After enlarging the wound of entrance the cap was easily removed with a small strabismus hook and a pair of forceps. The lid segment was still in it.

Improvement was prompt and uninterrupted, and promised to be complete until April 20th, when patient complained of vision in this eye getting dim. The vitreous was cloudy and contained numerous large floaters. This has gradually cleared up, and his vision is now equal to 20/20ths. With the ophthalmoscope one can still see a black pigmented area in the retina, on the nasal side near the periphery.

The copper cap and lid segment are here for inspection.

Discussion.—DR. ELLETT referred to a patient shown to the Society with a similar nonpenetrating injury to the eye from bird-shot. Subsequently the retina detached, and there is now complete atrophy of the iris and a blind and shrinking eye. The prognosis in these cases should therefore be very guarded.

Iritis and Cataract.

DR. E. C. ELLETT reported the case of Mr. M. A., age 72. Four years ago he had an unsuccessful cataract extraction on his right eye by another physician, and the eye was subsequently enucleated. Left eye incipient cataract. Vision 20/100 with glasses. Preliminary iridectomy Jan. 18, 1922. Not very long after this he had prostatic inflammation and an operation on the prostate; so it was the last of March before he returned for the cataract operation. This was deferred on account of a slight ciliary injection and he was sent back home with atropin. May 13th, pupil widely dilated, eye very red, spots of exudate on the posterior surface of the cornea and tension increased (62 McLean). Some improvement with rest, atropin, dionin and subcutaneous injection of milk. June 3rd, paracentesis. Since that time the eye has improved very much. Tension still increased (60 McLean).

Sclerocorneal Trephining.

DR. E. C. ELLETT also reported the case of Dr. L. H., age 66. March 3, 1922. Failing vision for two years, central vision 20/29 each eye, fields as shown on the charts. Eyes not inflamed. Pupil 5 mm. and sluggish, marked cupping of both discs. Tension mercury tonometer 25 and 29, McLean 50, each eye. The tension varied as shown on the tonometer charts. June 5th corneoscleral trephine with peripheral iridectomy on R. June 9th corneoscleral trephine with complete iridectomy L., 2 mm. trephine.

Epithelioma of Inner Canthus.

DR. R. FAGIN presented Mrs. N. G., age 28, from Covington, Tenn. For the past five years has been working as a "spinner" in the Tipton Cotton Mills. She claims that the cotton lint gets in her eyes very badly and irritates them. For the past three months, a small warty growth has rather rapidly increased in size at the limbus, toward the inner canthus of left eye. This growth has the appearance of an epithelioma. The patient is now in the hospital preparatory to having the growth removed. The vision in this eye is still 20/20. The growth when removed will be sent to the laboratory for diagnosis.

S. S. EVANS, Secretary.

WASHINGTON INTERNATIONAL CONGRESS.

Wednesday, April 26, 1922.

(*Continued from Page 761.*)

Vessels in Glaucomatous Patients.

DR. CARLOS CHARLIN, Santiago, Chile, had studied the cardiovascular conditions in 100 patients suffering from primary glaucoma; of whom 90 presented evidence of vascular disease. Of the 10 who were apparently healthy, 3 gave a positive Wassermann reaction.

Less than 30 years	1
31 to 40	2
41 to 50	26
51 to 60	30
61 to 70	31
71 to 80	9
Over 80 years	1

Of the 90 with manifest cardiovascu-

lar disease, 62 had arterial hypertension: 59 had aortic disease, of whom 21 showed lesion of the aortic valves; and 5 had myocarditis. The etiology of the vascular disease in 54 of these was attributable to syphilis; in 32 to arteriosclerosis, and in 4 to various or doubtful causes.

The study of the clinical material gathered by us teaches that the man suffering from glaucoma, in 90 per cent of the cases, is not alone suffering from his eyes, since he is also suffering from a general vascular affection. On the other hand, it is generally accepted to-day that in the glaucomatous process the fundamental anatomicopathologic lesion is the lesion of the ocular vessels. If both testings are compared, the general vascular lesion shown by the general clinical examination and the vascular lesion of the eye with hypertension, it is difficult not to accept a close relationship between the two processes.

At any rate, on account of the extreme frequency of a general affection with the glaucoma, we believe that these patients, after the ocular examination is made, should be submitted to minute clinical examination — inspection, auscultation, thoracic radioscopic examination, tests of arterial blood pressure, Wassermann reaction, and examination as to renal function. After the ocular and medical diagnoses are complete, there should be added to the ocular the general therapeutics of whatever condition the clinical examination may show.

Diagnosis of Glaucoma.

Dr. R. H. Elliot, London, England. Diagnosis helps us in two somewhat different directions. (1) To ascertain whether glaucoma is present or not; and (2) to enable us to decide whether to adopt therapeutic or operative measures in the treatment of individual cases.

There are few different problems confronting the ophthalmic surgeon which are more difficult than that of the diagnosis of a pathologic rise of tension in certain cases; yet a surgeon, who will take the trouble to avail himself to the full of the modern methods of diagnosis, need never be long in doubt as to the presence or absence of glaucoma. A correct diagnosis can sometimes be made only by waiting; but, if the surgeon is alive to the condition present, and loses no time in collecting the necessary data, it may be questioned whether the delay involved need ever lead to serious consequences. The same is true of the measures and observations necessary to enable us to come to a sure conclusion as to whether a glaucoma is progressive or stationary. Few systems are more evil than that of the collective consultation, in which a number of surgeons meet, conduct a superficial examination and assure the patient that he should or should not be operated upon. Such a method appeals powerfully to a patient and his friends. It is popular, dramatic and worthless. The diagnosis of a difficult case of glaucoma, whether the point at issue be the presence of increased tension or the question of the progress of the disease, can be established only by the work of an individual surgeon, who conducts a routine and careful examination to which he is willing to devote unlimited time. When all his data are available he may perhaps with profit lay them before one or more colleagues, and get the latter to check doubtful points which his work has revealed. This is not the method of the sensational novelist, the equally sensational newspaper paragraph writer, or, only too often the friends who prefer the consultation of a number of surgeons; but it is the one which best subserves the interests of the patient.

With this introduction we now take up a number of points in turn

THE CONJUNCTIVA AND SCLERA.

Circumcorneal congestion, which may be of a very pronounced type, is a constant accompaniment of acute and subacute glaucoma, whilst enlargement of the episcleral vessels, and especially of those which perforate the tunic of the eye, is the rule in chronic cases. These vessels are veins: (1) They have the appearance and color of veins; (2) when cut across in the course of an operation their bleeding is typically venous; (3) when the lumen of one of these vessels is closed at each of its ends as far apart as possible, it not only fills up from either end indifferently when the pressure is released, but it actually fills from lateral feeder vessels, even whilst the

two ends are still blocked; (4) under high magnification with a corneal microscope and Gullstrand slit lamp, no pulsation whatever can be observed in any of these trunks.

THE CORNEA.

The steaminess of this membrane in acute cases, and the secondary changes in late cases are too familiar to need more than a mention. There are, however, two symptoms due to the steaminess of the cornea which deserve notice, viz., mists and halos. The great importance of these lies, not merely in the fact that they attract the patient's attention very markedly; but still more in that they serve as a very delicate test of the recurrence and duration of exacerbations of the disease. Those who are suffering from simple glaucoma, even with mild congestive intervals, may never be conscious either of mists or halos; but the victims of acute and subacute attacks need never be in any doubt as to the recurrence of their trouble, once they have been trained to employ this test.

It is important to distinguish two quite different sets of conditions under which mists and halos are observed, viz., (1) in the early mornings, and (2) at those times of the day or night when fatigue is most pronounced. The early morning symptoms are due to the failure during the sleeping hours of the pump action, described by Professor Thomson, and exerted on Schlemm's canal thru the action of the ciliary and iris muscles. Those which come on late in the day, as the result of fatigue, are to be attributed to vascular congestion and are frequently relieved by food or rest. They, in fact, constitute the incursion into the case of the vascular factor as an outcome of disturbances of the vasomotor equilibrium.

Mists of Vision.—It is important to remember that these are due in part to edema of the corneal epithelium and of the superficial layers of the cornea, and possibly also of the endothelium lining Descemet's membrane; a similar condition of the retina almost certainly plays an important part in the production of the symptom. Such a condition is predisposed to by the cutting off in some measure of the arterial blood supply, and by the obstruction to venous return, both being factors which interfere with the due nutrition of the retina.

Halos.—These are best seen when looking at bright lights in the dark. When they are present, the ordinary patient can see them excellently around a match or candle flame held at arm's length. In the dark room the glow of a self lit ophthalmoscope bulb, especially when not too bright, gives very vivid halos. It is most important that our patients should understand what we mean by halos, since so many conditions may easily be confused with the true colored rings seen by the glaucomatous subject. For this purpose a glass plate dusted with lycopodium should be kept handy; once a patient has looked thru this he knows for all time exactly what we mean when we ask him whether he has seen colored rings.

The following conditions are mistaken for halos: (1) The golden haze made up from radiating luminous beams which a normal eye may see surrounding a light in the dark, and which is often dissipated by rubbing or drying the eye. (2) Imaginary halos seen by nervous people who have heard glaucoma discussed. (3) The scintillating scotoma of migrain. (4) The rapidly closing in rings of color seen by nervous or over tired people, when they shut their eyes in the dark. (5) The halos due to damage to the corneal epithelium produced by caustic or other medicinal applications. (6) The transient halos complained of by those who suffer from conjunctivitis. These are associated with mucous discharge, and they at once disappear on washing the eye; they are all probably due to the inclusion in the sticky mucus of masses of lymphocytes imprisoned in the conjunctival exudate. (7) The halos seen when looking at a bright light far back in a big room thru a steamy plate glass window from the dark outside; these are the same as those seen thru breathed on or steamy spectacle glasses.

(8) The halos seen by some patients in the early stages of cataract; these are said to disappear if looked at thru a pin point diaphragm. (9) The physiologic halos which any normal eye can see if carefully looked for. These are said to be brighter and more prominent in the old. (10) The physiologic halos seen by many normal eyes when the pupils are dilated.

The majority of these conditions can be easily differentiated from the glaucomatous halos by any surgeon who is acquainted with the fact that they may possibly be seen by non-glaucomatous people. A few inquiries will soon show their true nature. There are, however, some points which may here be emphasized. (1) The real glaucoma is much brighter than its imitators; the patient can very easily recognize at least three colors, a central blue, a middle yellow, and an outer orange red, or orange. In addition a band of green can often be detected between the blue and yellow. (2) The diameter of the glaucomatous halo varies considerably in different patients, and even in the same patient at different times. I have never found it less than 6.5 degrees, nor more than 11.54 degrees. The physiologic halos on the other hand, are stated by authorities to measure constantly about 7 degrees in diameter, and the colored rings are very feeble, requiring close attention for their recognition. (3) The fact that a patient sees a colored halo after the use of a mydriatic may easily lead to the suggestion that the surgeon has been guilty of negligence in the use of the drug. The danger is all the greater, since many members of the public are better informed on the subject of glaucoma halos than the majority of the medical profession. It is therefore essential, that in every such case we should take note of the diameter of the halos, of their brilliance, of the colors recognized, and of the tension of the eye at the time. By doing so we may save a colleague much underserved discredit.

So important did this subject appear to the writer, that he sought the assistance of Mr. H. H. Emsley, of the Northampton Polytechnique Institute, Clerkenwell. This gentlemen has most kindly spent a great deal of time and trouble over the matter. He points out that Druault's test, as suggested by Morax, is not quite accurate. If a true glaucoma halo, or any other halo due to interference with the corneal tissues, is observed and at the same time a straight edged screen (a stenopaic slit held vertically, does excellently) is moved across the area of vision close to the eye, the ring of the halo is obliterated just in proportion as the eye is covered by the slit. If, on the contrary, we observe a physiologic halo, and this is much easier with a dilated pupil, and move our straight edged screen across the area of the field of vision, an entirely different phenomenon is seen; due to the fact that the halo is here produced by the grid like crisscrossing of the lens fibers. As the screen comes across the eye, instead of seeing the complete halo, we now only see two rather narrow spectral bands, one at each end of a diameter of the halo. With the movement of the screen these swing around like the spokes of a cart wheel, traveling clock-wise or counter-clockwise alternately, according as the screen is moved in one or the other directions.

This beautiful little experiment of Mr. Emsley enables us to tell at once whether the halos—whether seen spontaneously by the old, or as the result of mydriasis in younger people—are produced by the lens fibers, or are truly corneal. In the former case they have no connection with the intraocular pressure; in the latter they may or may not have such a connection. A last word as to the measurements of halos: With the light ten feet from the observing eye, the diameters of the halos will be approximately as follows: For 4 degrees, 8.5 inches; for 5 degrees, 10.5 in.; for 6 degrees, 12.5 in.; for 7 degrees, 14.75 in.; for 8 degrees, 17 in.; for 9 degrees, 19 in.; for 10 degrees, 21 in.; for 11 degrees, 23.33 in.; for 12 degrees, 25.35 in.

THE ANTERIOR CHAMBER.

Shallowing of the anterior chamber may occur under two quite different conditions: (1) It may be the result

of an overdistension of the vitreous body, of congestion of the ciliary body, or both; or (2) it may result from the sealing down of the angle of the chamber, due to the adhesions of the iris base to the corneal periphery.

In either case, and especially in the latter, it may be important to be able to measure the depth of the anterior chamber. Much information may thus possibly be obtained as to the progress a case is making on the downward road, and as to the prospect of an operation proving a success. With regard to the latter point, the writer holds strongly that an iridectomy is useless once the angle is sealed down, unless it accidently gives rise to a filtering scar. If this be accepted, it is obvious that we should aim at performing a filtering operation to start with, and should not leave our main object to be the sport of chance. Again, if we desire to trephine, it is of importance to know beforehand how far forward the obliteration of the angle has progressed and, if we have reason to believe that it is a very far forwards, we should get all the corneal splitting we safely can.

Messrs. Zeiss have fitted to their corneal microscope a device known as the "Ulbrich drum." With the aid of this, and by a simple calculation, the depth of the anterior chamber can easily be measured. Even apart from any calculation, the relative depths of the same chamber can be estimated from time to time. This is probably the most important element in the case. These considerations have been put forward because it seems probable that important advances can be made if such lines of work are followed up; and America is far more likely to do the work than most parts of Europe, for the present at least.

Cloudiness of the anterior chamber, or deposits on the anterior surface of the iris, or posterior surface of the cornea, are signs of an iridocyclitis. Their observation is most important, as the diagnosis of the cause of the glaucoma, and the main indication for the exhibition of mydriatic drugs, hang on their recognition. This point was well brought out in the valuable discussion before the American Ophthalmological Society, at their 55th annual meeting, in 1919, in Atlantic City.

Koeppe has claimed that glaucoma can be recognized even in what he terms the "preglaucomatous stage," by the aid of the Gullstrand slit lamp, even months or a year before any other sign of the disease has appeared. He states, that as a result of the morbid changes, which are taking place, pigment granules are set free; and that these wander out in the form of a fine dust, and are to be seen on the surface of the iris stroma. The observation has been both supported and contradicted. The writer has certainly seen such granules in glaucoma cases. He has also seen unquestioned cases of established glaucoma, in which no trace of them can be detected. The matter is one of great interest. If we could, by any means, anticipate a certain diagnosis of progressive glaucoma, it would be of incalculable benefit to us, for we could then undertake a well planned operation at a date when everything is in favor of success, instead of being forced to resort to surgery at a dangerous time, as is now only too often the case.

THE CILIARY BODY.

The impairment of accommodation which accompanies glaucoma is too well known to require more than a passing mention. The appearance of a manifest hyperopia is less widely recognized as a sign of the disease, altho it is far from being uncommon. Its genesis is easily explained. The original condition in such cases was one of latent hyperopia; under the paralyzing influence of the increase of pressure, acting upon the third nerves and on their terminals in the ciliary muscles, the hyperopia has become manifest.

THE LENS.

Much has been written recently on the subject of cataract complicating glaucoma. It is necessary to distinguish sharply between 3 conditions which are often confused with each other:

(1) The form of cataract secondary to glaucoma is characterized by a want

of definition in its appearance. It looks like a smoky-greenish or bluish haze, and does not present the definite features with which we are all familiar in an ordinary primary cataract.

(2) Glaucoma secondary to cataract. The appearance of the lens in the majority of these cases is characteristic of the intumescent variety; the history is unmistakable, and the presence of a primary and hitherto uncomplicated cataract in the opposite eye clinches the diagnosis.

(3) Cataract, occurring as an accidental complication of glaucoma, is by no means uncommonly met with by any surgeon in large practice.

The socalled "green reflex" of glaucoma is not characteristic of the disease. Often it can be seen in old eyes; the conditions that favor its appearance are a dilatation of the pupil, combined with some want of perfect transparence of the aqueous humor, lens and cornea, or all three combined.

THE OPTIC DISC AND RETINA.

The typical *cupping of the optic disc*, on which so much reliance is placed, is a feature of established glaucoma, and is often absent in early cases. The sign may therefore fail us in the very class of cases in which the greatest difficulty in diagnosis occurs.

The method of formation of the cup in the great majority of cases is typical and unmistakable: (1) If a case is watched from the first, a slight and subtle change is observed at the very margin of the disc. Either around a large area, or thruout the whole circumference, it shows a slight depression, not sufficient to be estimated by the aid of the ophthalmoscope, but quite enough to be recognized by any trained observer. (2) At a slightly later stage a faint bend can be observed in the course of the vessels as they pass over this depressed margin. By this time the whole circumference of the disc tends to be involved. (3) As time goes on, the depression in the floor of the disc increases so it can be estimated ophthalmoscopically, and the kinking of the emerging vessels becomes increasingly accentuated.

The aberrent types of cupping are comparatively rarely met with. This is all the stronger reason for the surgeon to be on his guard and prepared to recognize them early. They are of two types: (1) The circular, central, physiologic cup, which gradually widens out toward the circumference on the papilla, and (2) the steadily enlarging physiologic cup. It is necessary to repeat that these are rare forms of cupping, and that the diagnosis of glaucoma in such cases must rest on a comprehensive survey of all the evidence that the case can afford. It is commonly believed that retinal *venous pulsation* is a sign of glaucoma. This view has recently been challenged, and the writer has therefore studied the subject very closely. There can be no question that venous pulsation, even strong venous pulsation, may be met with in eyes in which the tension is normal; and again that a complete absence of pulsation may be found in high tension eyes. There are, however, certain items of evidence which we must consider: (1) The exertion of a moderate amount of digital pressure on the globe will, in many cases, provoke a marked venous pulsation or exaggerate a pulsation already present. (2) The instillation of a mydriatic into the eye will, in a certain number of cases, have a precisely similar effect. (3) In a number of cases of glaucoma, a very marked venous pulsation will be found present, and in not a few of these this pulsation will disappear, or greatly lessen in character, after the performance of a successful decompression operation.

The writer's view is that when the other features of a case point toward glaucoma, the presence of a marked venous pulsation in the retina is a suggestive piece of contributory evidence. Further, the presence of such a marked venous pulsation, whenever it is detected, should raise in the observer's mind the possibility of a threatening of glaucoma. The circulation within the eye has a marvelous power of adapting itself to altered conditions, but in the transition stage from one condition to another the vascular system may give such evidence as we have been dis-

cussing, of the strain which is being put upon it.

Spontaneous pulsation of the *retinal arteries* is spoken in the textbooks as tho it were common, if not an invariable accompaniment of glaucoma; whereas the truth is that it is a sign which is very rarely seen indeed, and then only in the course of congestive attacks of the disease, passing away as soon as the exacerbation is got under control. The writer cannot recall a single instance in which he has detected it in an eye suffering from simple glaucoma, or in a congestive case in the interval between two attacks. In many of our cases of acute glaucoma, and by this we mean exacerbations of a glaucomatous condition, the media are too hazy to allow us to see pulsation of the retinal vessels. In others we can see the vessels and note the absence of pulsation, whilst in still others (and these are very few in number) a distinct and sometimes a very marked arterial pulsation may be clearly discerned.

We know that arterial pulsation means an intermittence of the even flow of blood thru the arteries, as a result of the intraocular pressure having risen to such a height that it is greater than the retinal arterial pressure, during the diastolic phase of the latter. In other words, it is clear evidence that the retinal circulation has become intermittent instead of continuous.

Very different grades of this condition can be recognized: (1) The arterial pulse is nothing more than a flicker, which affects the whole course of every branch of the retinal artery on the disc and even beyond it; it can be counted against, and confirmed by the radial pulse. Here we obviously have to do with the slightest possible interference with the retinal arterial circulation; it is only at the lowest phase of diastole that the even flow of the current is interrupted. (2) The arterial pulse becomes more and more marked; the phase of blanching of the artery may be so prolonged as to equal or exceed that of its greatest fulness with blood. Here the interruption of the retinal circulation is obviously becoming very serious. (3) In extreme cases the intraocular pressure has become so excessive that circulation can only be maintained at the height of systolic pressure; the phases of blanching of the vessels exceed those in which they are filled; giving almost the appearance of an aortic pulse, and justifying the similie used by a French writer; that the blood seems to spill in jets over the edge of the disc.

It will require but little consideration to show any thoughtful mind, that the watching of this phenomenon will provide valuable clinical data to observant ophthalmologists. So far we have been discussing the spontaneous arterial retinal pulse. We shall now turn to consider a phenomenon which can be observed not only in glaucomatous, but also in healthy eyes, and yet one which has a very definite clinical significance.

The induced *pulsation of the retinal arteries.* It is well known that by the exertion of digital pressure on the globe of the eye we can produce a pulse in the retinal arteries. This pulse follows closely the characters described in dealing with the spontaneous arterial pulse. With mild pressure the phase of interruption is extremely short. As the pressure is increased, it lengthens until it exceeds the period of onward flow. Finally if the pressure is made still greater, and it is questionable whether we have the right to employ such a means, the whole circulation thru the eye is stopped, and the current thru the retinal arteries definitely ceases.

The real value of these observations lies in the fact that, as the intraocular pressure rises, the amount of digital pressure necessary to produce a retinal arterial pulse tends to become less and less. Consequently, when we can evoke an arterial pulse by light pressure upon the eye, we are led to the conclusion, that the intraocular pressure has risen until it is nearly equal to the diastolic arterial pressure. The word "tends" has been advisedly and deliberately used, and for the following reason: There are cases of undoubted glaucoma in which quite considerable pressure is required before

the arterial pulse can be elicited; and there are cases in which no rise whatever of intraocular pressure has taken place, in which the diastolic pulse can be very easily produced by light pressure. This latter occurrence is most often met with in young persons. It is obvious that the former class of cases, the arterial pressure has risen *pari passu* with the rise in intraocular pressure—one more instance of the compensatory mechanism which is to be found in the eye, and of whose working we understand so little. In the latter class we appear to have to do with a condition in which the normal diastolic arterial pressure is unusually low.

Pallor of the Disc.—This pallor may be found under two quite different conditions: (1) It is seen in some cases of glaucoma at a very early stage, and is then presumably due to a constriction of the vascular supply as a result of the increased ocular pressure. That this should be so will surprise no one who is familiar with the very marked blanching of the disc, which is produced by digital pressure. It has been claimed that it may be a very early and very suggestive sign, in the absence of other evidence of an increase in intraocular pressure. This is certainly a point which should be carefully kept in mind, for in no disease is it truer that every shred of evidence helps. (2) It is met with as a marked, and often as an unmistakable, feature of established glaucoma. Its interest here lies not so much in establishing the diagnosis of the disease; which by this time has usually already been made without difficulty, but in furnishing a criterion as to the probable prognosis for vision, after a successful decompression operation has been performed. A similar relative pallor of the retina may sometimes be observed. This is, however, very difficult to appreciate, and a more significant sign is therefore to be found in a slight relative decrease of the diameters of the arteries as compared with those of the veins.

A dragging over of the retinal vessels in a bundle toward the nasal side of the fundus is a not uncommon phenomenon in late cases of glaucoma, and the appearance presented is very striking. Even when there are considerable opacities in the media of the eye, the bundle of red lines running inwards, and the contrasting dead whiteness of the disc in every other direction, furnish a picture which is unmistakable. This dragging over of the vessels is probably always associated with some measure of posterior staphyloma.

The Size of the Globe.—According to Priestley Smith, the average horizontal diameter of the cornea is 11.1 mm. in the glaucomatous as against 11.6 mm. in healthy eyes. The importance of this fact lies in the help it gives us when patients, with a glaucomatous family history, consult us as to the condition of their own eyes, being made nervous by the fear that they may have inherited the disease. Should we find that they have a low corneal diameter, we would be justified in recommending them to keep themselves under periodic observation, and to report the appearance of any suspicious symptoms of the disease. On the other hand, the observation of a large corneal diameter would be a distinct factor in enabling us to give a good prognosis.

The writer has been extremely dissatisfied with the devices employed for making these measurements; they seem to him far too rough and ready for their purpose. If it is imporaant to have the information, that information should be as exact as it is possible to make it. This is the principle on which all our examinations of the glaucoma patient should be conducted, if the opinions we give are to be really reliable. The requisites for a perfect instrument of the kind are: (1) A headrest must be provided so that the patient may be absolutely steady. (2) The instrument itself must be rigidly fixed on a firm stand. (3) The observed eye must have a definite point of fixation, so that it may not make the least move during the examination. (4) The scale and the eye must be sufficiently magnified to enable the observer to measure accurately in tenths, and preferably in twentieths of a milli-

meter. Messrs. Zeiss are constructing such an instrument especially for the writer, which will give a magnification of 8 diameters. This can be substituted for their corneal microscope on the standard adjustment stand of that instrument, and will be focused by a rack and pinion movement.

SUBJECTIVE PHENOMENA.

Pain.—Speaking broadly, pain is here as elsewhere, a measure of the congestion present. A simple glaucoma may run its whole course, without one moment of pain in the eye. Again, the early slight attacks of subacute glaucoma may be attended only by mild feelings of discomfort or of ocular pressure. On the other hand, the acute disease is marked by great suffering. Our principal interest in the subject lies in the fact that the trouble is not always referred to the eye. The patient, and even his medical advisor, may make the mistake of thinking that the cause of the trouble is in the teeth, the ears or nose; since the "neuralgia" complained of appears to start from one of those organs. Again as we well know, the headache, pyrexia and vomiting may lead to the erroneous and mischievous diagnosis of that refuge of incompetence, "the bilious headache." The taking of a meal or the obtaining of sleep may greatly alleviate the symptoms; such drugs as phenacetin and antipyrin are useless, while the bold instillation of miotics acts like a charm. All these are points that help the careful surgeon to a right appreciation of the cause of the patient's suffering.

Lacrimation.—According to Morax, various reflex troubles may suggest the commencement of glaucoma. The most suspicious of these is intermittent lacrimation, unconnected with wind or other provocative agency.

Nausea and Vomiting.—Morax states that, even if the glaucoma is untreated, the actual vomiting subsides within 24 hours, or at most 28 hours, while the sensation of nausea may persist for several weeks. He has met with patients suffering from subacute attacks, in whom the feeling of nausea was the only warning sign of a fresh rise in intraocular pressure.

Photopsiae are not unknown in the early stages of congestive glaucoma. They may occur as flashes of light, as the impression of a ball of fire which rolls across the field of vision, as sudden sharp, flash-like spots of light, or as a continuous luminous glow, lasting from seconds to minutes, or even longer. The patient is most apt to suffer from these photopsiæ when he is tired, and when he first gets into bed at night. It must be remembered that like symptoms may be observed under any conditions in which the retina is irritated, dragged upon, or otherwise interfered with. Nor must we forget that similar phenomena, but of central origin, are met with in neurasthenic patients suffering from errors of refraction, from migrain, and from other nervous troubles. Rest tends to relieve photopsiæ of retinal origin, whilst those due to cerebral troubles are often worst at night. Rainbows around lights have already been discussed.

Diminution of Visual Acuity is a frequent, if not a constant, sign of glaucoma. Its causes may be classified into medial and neuroretinal. The former includes interference with the refractile power of the cornea owing to overstretching of that membrane; corneal edema; deposits on the back of the cornea; degenerative and other permanent changes in the cornea; turbidity of the aqueous; opacities of the lens; deposits of various kinds on the surface of the lens capsule, and vitreous opacities.

Neuroretinal causes include pressure on and overstretching of the bundles of the optic nerve and of the layers of the retina, and starvation of the nerves and retina due to interference with the blood supply, as a result of intraocular pressure. The lesion may be manifested by a diminution of central vision or by an interference with the visual field. The former is practically always an evidence that a congestive element has entered into the case, otherwise good central vision is retained almost to the last. Defects in the field, on the other hand, are to be attributed to injury to the nerve fibers at the edge of the disc, reinforced by starvation of the retinal

blood supply. The distinction is obviously important.

The Visual Field.—The subject of the changes in the visual field in glaucoma is so vast, that it will only be possible to touch on a few of the main points of interest. The examination of a suspected glaucoma patient is very incomplete unless the fields have been carefully taken. Nor does one measurement suffice for each eye. The whole field should first be charted on a short radius (33 cm.) instrument, care being taken that the moving object is sufficiently small (not above 3 mm. in diameter for a daylight instrument). Next, the central portion of the field, lying within 26 degrees of the center, should be examined at a distance of 1 m. with a 1 mm. object. The results obtained by the two procedures are best kept on separate charts.

Perimetry of the Whole Field.—From this method we obtain very important indications: (1) There is a tendency for the nasal portion of the field to be affected before the temporal, and also for it to be more affected than the temporal, as the disease runs its course. Tho this is not an invariable rule, it is so frequent an occurrence that it deserves to be kept carefully in mind. (2) As the disease progresses, the whole field tends to shrink from the periphery towards the center. (3) The blind spot shows distinct signs of enlargement, even on examination by the small perimeter, if the fields are carefully taken from time to time as the disease progresses.

Roenne's step appears on the charts. This has been deliberately left until the last, not because it is the least important, but because the writer desires to draw special attention towards it. This sign is far too little known and appreciated. It is an evidence of the lesion of the optic nerve bundles at the edge of the disc, and it therefore clearly points to the essential pathologic process which is responsible for the harm done to the optic nerve as a result of increased pressure within the eye. This does not mean that it is necessarily pathognomonic of glaucoma, for we know hat other morbid conditions may damage the nerve fibers as the latter flow over the edge of the disc. Nor again, does it imply any doubt as to the influence of the vascular factor in the damage inflicted on the optic nerve and retina.

It is obvious that the nerve fibers and retinal elements which are starved of arterial blood and engorged with venous blood, as a result of increased pressure within the eye, must be thereby placed in a condition unfavorable to the resistance of trauma. In this we see the contributory element furnished by interference with the retinal and choroidal vascular blood supply; but it cannot be too strongly emphasized that for the essential lesions of glaucoma, and for the evidence whereby we diagnose them by means of perimetry, we must look to the edge of the disc and to the damage done to the nerve fibers at that area. This being so the importance of Roenne's sign stands out large against the background of other perimetric observations. Why has it been so little appreciated, and why is it so seldom observed.

The answer is: Because so much of the perimetric work in consulting rooms and other places is done against time, and sufficient leisure and trouble is not devoted to it. Moreover, as has been so often pointed out, this sign is best observed when the *circular* method of perimetry is adapted, for the simple reason that fibers whose damage we are studying run in arc like curves around the center of vision, and any lesions they sustain are most easily studied when the observed object travels along the length of the fibers instead of across them. To take 8 radial measurements of a field, and then to fill a chart by drawing lines between them is not perimetry; on the contrary it is bad work and is most unfair to the patient. It will not reveal the presence of a Roenne's step, nor will it give a true or even an approximately true idea of the state of visual field.

One point more deserves to be remembered: The glaucoma patient is sensitive to *changes in light*, and it is therefore important that periodical examinations of his field should be made as nearly as possibly under the same

conditions of illumination. It is difficult to obtain these by daylight in Northern latitudes, and for this reason self lit instruments, or those illuminated by artificial light are unquestionably preferable to daylight apparatus. At the same time it is very important to know the limitations of the instrument we use. These can only be learned by constant practice. Under no circumstances must either the fixation or the traveling object be too bright. If it is so the patient will soon become fatigued, whilst before this happens the limits of his field will be exaggerated.

Scotometry or Central Perimetry.—Bjerrum was the first to point out that in glaucoma we commonly meet with arc like scotomata which are connected with the blind spot, and which curve around the center of the field of vision to end on the horizontal raphé. Such scotomata may be met with above the horizontal raphé, or below it, or in both situations simultaneously. In the last cases we get ring scotomata. He maintained that these scotomata were the expression of lesions of nerve fiber bundles, at the edge of the disc. Roenne working on Bjerrum's hypothesis, looked for and found the sign, which is called by his name, and which we have already spoken of in connection with perimetry of the whole field of vision.

Seidel went a step farther and showed that very early in glaucoma, before any other sign of the disease could be certainly recognized, enlargement of the blind spot either upward, or downward or both, could be detected with certainty in a number of cases. These enlargements were always described as ending in single, pointed or rounded ends. From the first it appeared to the writer that this clinical feature was inconsistent with the accepted pathology of the condition, for, if it were a question of a lesion of a number of bundles of nerve fibers selected, as it were, out of the whole mass of the nerve, it would only be reasonable to expect that the lesions they would sustain would vary widely amongst themselves, and that therefore the scotoma produced would not end in a point but in a number of points. Experiments with Bjerrum's screen and with other apparatus of a similar nature failed to confirm this suggestion, until the writer devised the scotometer which bears his name. Then the scotoma with jagged points was at once found and has since proved to be so consistent a manifestation, as to make the appearance of this phenomenon of high diagnostic value.

Three principles are involved in the make up of this apparatus: (1) That of the circular, instead of the radial, method of examination of the field of vision, as advocated by Priestley Smith; (2) that of the magnification of the scale on which the phenomena are observed, so making the results easier to obtain and more striking to the examined eye, as advocated by Bjerrum; and (3) that of the examination of the field at intervals of 1 degree instead of those of 5 degrees or 10 degrees as is so often done. This last was a device which suggested itself to the writer's mind as likely to bring out the jagged nature of the scotoma, if this could by any means be accomplished.

It has been pointed out by some that the writer's sign is not obtained by other forms of scotometric apparatus, and it has consequently been suggested that it is an artefact. That this argument is not a very strong one is clear from the fact that the sign in question has been obtained in glaucoma cases by a number of reliable observers, who have abundantly confirmed the writer's findings with this instrument. A little consideration would show that it would be only natural to expect more accurate results from the Elliot instrument, when dealing with glaucoma cases, than from an ordinary scotometer; and this for the following reasons:

(1) As has already been shown the circular method has especial advantages in dealing with scotomata which are the result of lesions to nerve fibers at the edge of the optic disc, for these fibers sweep in curves around the central area of vision to reach the horizontal raphé; and it is always much easier, both for the surgeon and for the patient if, in taking a scotoma, we

pass thru its longest and not thru its shortest axis. Moreover, in the author's experience, a scotoma will always be carried further, and therefore shown at the greatest advantage, if we pass from the blind into the seeing area, instead of in the opposite direction.

(2) One can explore the whole field out to the 26 degree circle at 1 degree intervals by means of 26 circles easily, accurately and mechanically traced for us by a rotating disc; whereas to do this at the same intervals, working radially from the center, would demand 360 observations and would hopelessly tire our patients. Moreover the closely set radial lines for the first ten or fifteen degrees are practically impossible to dissociate from one another. (3) Whatever may be the verdict of other surgeons, nothing can shake the writer's conviction that the magnification of scotomata, whether these be physiologic or pathologic, makes the patient's task enormously easier, and the surgeon's results much more accurate.

The normal *blind spot* as mapped out by the new instrument does not differ materially from the records of other instruments employed for the same purpose. This is all the more important, since the field changes which it reveals in glaucoma are so very distinctive. We shall now consider these:

The jagged enlargement of the blind spot is so marked a feature in many cases, that it cannot easily be missed by any one who works with reasonable care. Even in quite early cases, the pointed enlargement of the normal physiologic scotoma may be highly suggestive of the presence of intraocular pressure. On the other hand, if under this test we find the blind spot of absolutely normal size in a suspected eye, we must regard this negative evidence as of great value in suggesting that there is no rise of intraocular pressure present, or at least that any such rise that may be, or may have been present, has probably up to date done no harm.

Detached Paracentral Scotomata.—A certain amount, tho not a great deal, has been written about scotomata in glaucomatous eyes, which are found at some little distance from the blind spot. Such defects are not infrequently met with when using the author's scotometer. If such a case is followed for some time, these defects can often be traced until they join up with the enlarged blind spot. They are obviously due to lesions of those nerve fibers, which are distributed to parts further away from the disc. Consider for a moment a bundle emerging from the optic nerve at the disc edge. It would naturally be expected that the fibers which would be earliest and most damaged would be those on the periphery of the nerve; such a lesion would be evidenced by an enlargement of the blind spot. If, however, those fibers which are distributed to the area of the retina, represented by that portion of the field close to the horizontal raphé, happen to be earliest damaged, we get one of these detached, paracentral scotomata close to the horizontal meridian. Again, if some of the intermediate fibers are damaged, we get a scotoma in corresponding position somewhere along the curved arc of the course of the bundle.

In explanation of these vagaries in the scotometric phenomena, the following considerations may be suggested: The bundles at the periphery of the nerve are most liable to be damaged by the sharp edge of the scleral ring over which they curve to reach the retinal surface. The central bundles, destined for the more peripheral parts, are more likely to be injured by the overstretching to which they are subjected when the nerve is pushed back under pressure. Variations in the anatomic arrangements of the nerve fiber bundles themselves may possibly explain some of the discrepancies in the phenomena observed. There are considerable anatomic variations in the supporting framework of the optic nerve head, and it is highly probable that consequently the nerve head yields differently in different eyes, and unevenly in the same eye. We are therefore justified in expecting that the injury inflicted will vary from case to

case, and will materially so influence the signs of disease presented by the perimetric picture.

(3) Invasions of the central area of the field. The surgeon who employs the writer's scotometer as a routine step in the examination of his glaucoma cases will be surprised to find how often the central area of the field is affected in eyes in which, under an ordinary superficial examination, he would not have suspected any such defect.

This is a matter of great interest and importance. The writer has seen patients whose medical men believe that they are holding their own, and whose fields, taken on an ordinary small radius perimeter, supported such a view, and yet they themselves were firmly convinced that they were losing ground, as indeed they were; careful scotometry at once showed that they had good reason for their complaints. The value of the writer's instrument in explaining such cases, in following doubtful eyes before coming to a decision as to operation, and in gauging the effect of operative or other treatment must be experienced to be appreciated.

The invasions of this area of the field tend to move steadily onward toward the obliteration of the upper, or of the lower central field, or of both. Two clinical types are thus evolved: (1) When the scotoma is confined to either the upper or the lower field, the condition may amount to a nearly complete central hemianopia before the patient is aware of the serious defect in his vision. This can only be the case when the opposite eye is comparatively normal. For obvious reasons the hemianopia is always superior or inferior, and never lateral. (2) When the invasion of the central field takes place simultaneously above and below the horizontal meridian, the patient is more likely to detect it at an early date. The progress of the formation of a ring scotoma can be watched in such cases thruout its stages.

The diversity met with in different glaucoma fields has already been commented on, and some indications of its various causes have been furnished. We must now shortly discuss the influence of the incursion of the vascular factor on the broad features of these fields. As has already been pointed out, the field changes which are due to the mechanical effects of intraocular pressure owe their variations to anatomic differences in the supporting framework of the nerve head, and in the arrangements of the fibers of the nerve. If the congestive element could be excluded entirely from a case, we should get very different, but always clear cut, perimetric pictures, and from these we would be able to speak with very little hesitation as to the nature and extent of the damage which has been inflicted on the optic nerve.

This, however, is far from being the case. The element of congestion has to be reckoned with in most instances. (1) In some it dominates the whole picture, blurring all details like a fog lying across a landscape. (2) In others it modifies our findings to a considerable extent, and yet we may be quite unable to apportion the just amount of blame to the two great factors, the mechanical, modified by anatomic circumstances, and the congestive. In yet others, the vascular factor is so weak that it is with the utmost difficulty we can trace its influence. This, however, we may say truly, that while simple glaucoma tends to produce definite and uneven curtailments of the field, the entry of the vascular factor blots out, or tends to blot out, the sensitiveness of the whole retinal area.

TONOMETRY.

There are comparatively few ophthalmic surgeons, and probably none in America, who do not make abundant use of one or another form of tonometer. Even the student of to-day is trained to employ this instrument just as naturally as he does the thermometer or the binaural stethoscope. The ophthalmologist who does not do so, fails in his duty to his patients and is an anachronism. Some use the Schiötz, some the Gradle and some the McLean. It matters comparatively little which model we adopt, so long as we keep to one and learn to understand

its indications. Nor is it a matter of any great importance from the clinical point of view, that the translated reading in mm. Hg. may not accurately represent the intraocular pressure in the eyes measured. We cannot fix for any tonometer a point below which the recorded pressure is certainly normal, or a corresponding point above which it is supernormal. For we know that the pressure may vary considerably in different normal eyes; what may be low for one may be normal for another, and what may be normal for a third may be high for a fourth. It is to take an altogether perverted view of the use of a tonometer to regard its readings as if they were of the nature of a scientific laboratory experiment. They are nothing of the sort, and it may be a long time before they ever become as accurate as this.

On the other hand, the indications they do give us are of the greatest possible value: (1) In a very large number of cases they tell us most definitely whether the intraocular pressure is distinctly high or normal. (2) When the pressure is different in the two eyes and especially if there are other indications of glaucoma in the higher tension eye, we are confronted with a very strong suggestion that a pathologic rise in pressure is present. (3) Variations in the pressure of an individual eye occurring from time to time, afford us a most valuable criterion of the progress, favorable or otherwise, that the case is making, and of the value of the medical, operative, or other means that we are employing in the treatment of the case. In this connection Butler's tonometer charts deserve a mention, as they enable the surgeon to follow up his cases at a glance.

It must be quite clear that nothing which has been written above is meant as a criticism of the efforts which have been made to standardize the Schiötz tonometer. Such an aim is altogether desirable, and I would like to pay here a tribute of admiration to the splendid work done in this field by American surgeons and especially by McLean.

THE LIGHT SENSE.

There can be no question that the light sense is profoundly affected in glaucoma. We know this well from the bitter complaints of our patients. They frequently tell us of their difficulties when they pass from light into darkness, or vice versa. Moreover, bright days dazzle them, whilst in dull cloudy weather they have difficulty in finding their way about. An examination of the light sense ought therefore to be productive of valuable data.

It has been claimed by reliable British writers that in the earlier stages of glaucoma there is a rapid reduction in light minimum sense, but only a very slight reduction in the light difference sense, whilst in incipient atrophy the reverse is the case. On the other hand, French writers have obtained diametrically opposite results, finding the light difference sense the first to be attacked, and the light minimum sense to be only diminished in the presence of optic atrophy. The writer has been much troubled over this question. He has met with well established cases of glaucoma in which the light sense, tested by various instruments, does not appear to be inferior to the normal, either in minimum or in difference, and yet the patients undoubtedly suffer when the light is reduced. He suggests as a possible explanation that the central light sense, like the central visual acuity may long remain practically normal, whilst the peripheral light sense may share the deterioration with which we are so familiar at the boundaries of the visual field. The writer would emphasize that he in no way wishes to set up his own opinions on the subject against those whose findings differ from his, and that the remarks he has now made on the subject are merely a call to other workers to persist in unraveling a tangled skein. Nor is the interest of the subject purely academic; on the contrary, the most valuable results from the point of view of the early diagnosis of the disease may spring from such work. It would be of interest if members of the Congress would give their views on the best pattern of photometer for use in the examination of glaucoma patients, and if they would at the same time, indicate the means they employ for differ-

entiating central and peripheral acuity of vision for light.

CONCLUSIONS.

This paper has endeavored to take up and deal with a few of the less well understood aspects of the diagnosis of glaucoma. The subject is far too vast to be treated at length, and the writer is deeply conscious of the many deficiencies of his contribution. There are certain points that he would like to insist upon with the utmost emphasis:

1. In order to decide whether glaucoma is present or not, the patient should be exhaustively examined. This is a long, painstaking, fatiguing business, and cannot be accomplished at one sitting, or the subject will be over tired, and the data collected will be untrustworthy. Given time, patience, and suitable equipment, any surgeon should be able to make up his mind definitely either (1) that a patient has glaucoma and requires treatment or, (2) that he has not got glaucoma; or (3) that, in the absence of definite and distinct signs of the disease, the case should be followed and watched until an unhesitating opinion is arrived at.

No means should be neglected to make the diagnosis as precise as possible; the history should be taken; a routine inspection of the eye should be made in a good light without any form of apparatus, and later an examination by oblique illumination with the aid of a corneal loupe, the refraction should be carefully estimated; the normal diameter and the depth of the chamber should be measured; further details should be sought for with the corneal microscope, the fields being illuminated by a Gullstrand lamp; the possibilities of ophthalmoscopy, perimetry, scotometry, tonometry and photometry should be exploited to the full; halos, if present, should be measured. Then if the surgeon, having thus made the most of the means at his disposal, is still in doubt, he has little, if anything to lose by watchful waiting.

2. The second point is the complement of the first: A diagnosis of glaucoma should never be made on any one sign or symptom, no matter how suggestive it may be. Such a course can never be necessary and is not justifiable.

3. Once glaucoma has been definitely diagnosed, the patient should be very carefully watched, and if he is going down hill in spite of general and therapeutic treatment, an early operation should be undertaken to reduce the intraocular pressure. The recurrence of congestive attacks is a strong indication for a decompression operation. One of the most delicate tests of such recurrences is the observation by the patient of halos around lights. If the congestive attacks are well marked, the diagnosis is obvious. It is only in the very mild subacute exacerbations of glaucoma that we need to rely on such a test.

In these cases where all evidence of congestion is absent, we must look for our indications to scotometry, tonometry, ophthalmoscopy, the testing of central visual acuity, etc. The order is deliberate, and indicates the writer's views as to the relative importance of the methods.

Much has been said and written about an exaggerated tendency to resort to operation for glaucoma. What has impressed the author more than anything else in connection with this disease, during the eight years that he has practiced in Europe, has been the inclination, even of very able surgeons, to postpone a glaucoma operation if it is possible to do so. He believes that where one operation is undertaken too early very many are put off until far too late. From the days of von Graefe and de Wecker onward, it has been a guiding surgical principle that the earlier an operation is undertaken for the relief of glaucoma, the better is the prospect of success. It is the operations performed at a late stage which give us the worst results, and the later the stage the worse the prognosis.

Blood Pressure in the Vessels of the Eye.

DR. A. MAGITOT and DR. BAILLIART, Paris. It has long been known that a slight compression of the eyeball causes arterial pulsation in the retina. Physiology enables us easily to understand that this arterial pulsation ap-

pears when the tension of the ocular media reaches the diastolic pressure of the central artery of the retina. Similarly, we know, that the pulsation will cease when the pressure exercised thru the ocular media is greater than the systolic pressure of the central artery. It was soon discovered that, in the cases of patients suffering from arterial hypertension, it was necessary to exercise on the eyeball a strong pressure to bring about the disappearance of the retinal pulsation; Bailliart (1909), then Melville Black (1911), and Deyl (1912) had drawn attention to this method of recognizing the existence of arterial hypertension.

In order to measure the arterial pressure in the central artery of the retina, it remained therefore only necessary to know the amount of force it was necessary to apply to the eyeball to bring about the appearance and disappearance of the pulsation. Th. Henderson had, for this purpose, constructed as early as 1914, an apparatus which calculated in millimeters of mercury the pressure which, exerted on the exterior surface of the eyeball, was sufficient to bring about the appearance of the diastolic pulsation. Henderson thought, which is not altogether incorrect, that by adding the ocular tension to the figure given by his apparatus, he would get the diastolic pressure of the central artery.

Bailliart in 1917 suggested a method for determining the figures of the diastolic and systolic pressures of the central artery. By means of a special dynamometer, graduated in grams of water, so that it might be easily verified on any scales, a pressure is exerted on the eye in the region of the insertion of the rectus externus until the operator notes the appearance and then the disappearance of the retinal pulsation. Two successive readings will thus give the amount of force which it is necessary to exert to balance the diastolic and then the systolic pressure of the central artery. But the thing that it is then important to know is, how the primitive ocular tension has been modified by these dynamometric pressures; for, finally, it is by its intermediary that we act on the vessels of the eye.

Thomson Henderson has had the idea of measuring in millimeters Hg. the pressure exerted by him on the eyeball and of adding to it the ocular tension determined by means of Schiötz's tonometer. But in this method there is a double cause of error; first, from a physical point of view, two successive pressures do not add up together; and on the other hand, the pressure transmitted by a liquid is proportional to the surface compressed. Here is, however, another method:

In a dark room, the patient being placed in a recumbent position, the operator causes the appearance and disappearance of the pulsation and notes the weight in grams necessary to obtain these two results. Then Schiötz's tonometer is applied to the eye, while at the same time by means of the dynamometer is exerted a pressure equal to that which was found necessary a few moments before to produce the appearance and disappearance of the pulsation. The tonometer weighted with its heaviest weight will indicate approximately in millimeters Hg. the pressure exerted on the vessels of the retina. It is evident that the greater the pressure exerted on the eyeball the greater will be the ocular tension.

But this method complicates the operation, and is only possible with exceptionally docile subjects. It is for this reason that we undertook to study experimentally, on an animal, the modifications of the intraocular tension under different pressures, and to draw up a chart. But to be of use clinically, the knowledge of these modifications of the "ophthalmotonus" caused by pressure exerted on a healthy or diseased eye, must apply not only to a particular case but to a whole series of figures of initial tensions. The method would indeed be singularly restricted, if we only knew the tensions produced by a pressure of 10, 20, 40 or 80 grams on an eyeball, the initial ophthalmotonus of which would be uniformly 20 mm. Hg. It is indispensable to acquire a knowledge of the effect produced on different initial ophthalmotonus, for we must evidently suppose that the same pressure of 20

grams will produce a different tension in an eye of which the primitive ophthalmotonus was 15 mm. Hg., and in another of which the ophthalmotonus would be 30 mm. Hg.

The drawing up of such a chart was possible only by experiment. We carried our researches on cats anesthetized with chloralose, this product being easier to manipulate than curare, and causing only a slight modification in the general blood pressure, on condition that the animal is not allowed to get cold. We have also made use of a mercury manometer invented by one of us, the manipulation of which appears to us to be easier than that of other instruments.

The ocular tension of the cat oscillates between 15 and 25 mm. Hg. according to conditions. It was therefore relatively easy to establish the figures of ophthalmotonus starting from these initial tensions. But it was necessary to obtain greater tensions and others still weaker. We were able to produce higher starting figures by practicing subconjunctival injections of NaCl. We were thus able to obtain quite easily tensions of 40 or 45 mm. Hg., rarely more.

In order to operate on lower ocular tensions than 15 mm. Hg., we had recourse to a deeper narcosis in order to act upon the general blood pressure. But we were unable to obtain less than 10 mm. Hg., which in the cat as in man is the average postmortem tension.

(1) We chose the cat in preference to the rabbit, which is, however, an easier animal to handle, on account of its ocular nervous system which is very similar to that of man. Yet both of them possess a much larger anterior chamber, and we do not know whether the general elasticity and resistance are identical with those of the human eye.

We have avoided producing modifications of the tonus by injection or aspiration of the intraocular liquid, as these means give erroneous figures for the two following reasons:

1. On account of the resorption.

2. On account of the intraocular vasodilatation, which very probably takes place after a few seconds. As to the pressures, they are made by means of Bailliart's dynamometer, which is either held in the hand or immovably fixed in a ratchet support.

The accompanying chart will avoid any useless descriptions. It gives us the figures for pressures applied horizontally (as on a patient sitting or reclining) on eyeballs, of which the initial tensions vary from 10 mm. to 50 mm. Hg. It will be noted that the different lines starting from different initial tensions, are remarkable for their parallelism and also for being nearly straight. It results from this, that this chart can be completed for all the initial pressures and for all pressures applied.

It will be seen that with this chart the determination of diastolic or systolic arterial pressures in the branches of the central artery becomes easy.

We first measure with Schiötz's tonometer the initial ocular tension, then we seek what dynanometric pressure will bring about the appearance and later the disappearance of the arterial pulse. We then refer to the chart and find in millimeters Hg. the required figures. In an eye of normal tension, in a subject whose arterial pressure is normal, the arterial retinal pressure is about 35 mm. for the diastolic and 70 mm. for the systolic.

If the retinal circulation has a primordial importance since it insures the visual function, the choroidal circulation also plays a considerable part; the part it takes in the modifications of the ophthalmotonus is well known. It is, moreover, possible by studying the effects produced by compression of the eye, to obtain notes on the uveal circulation similar to those obtained on the retinal circulation.

Sometimes, particularly in extreme myopia, it is possible to perceive thru the retina the choroidal vessels; but we can but ill distinguish the arteries from the veins, and on the other hand these vessels never show unless in certain exceptional cases, either spontaneous or forced pulsations. When considerable pressure is applied, it is even difficult to tell at what precise moment they are emptied, and even if we did know it, this would have little value since we cannot distinguish the small arteries from the small veins. Conse-

quently the direct examination and measuring of the arterial pressure in the choroidal vessels is not yet possible.

In a recent work, G. Leplat has shown that it was possible, under certain favorable conditions, to study by vision the blood pressure in the arteries of the dog's iris, by making use of the process we have described. The importance of this fact is truly considerable; this process enables us to acquire for the first time some notions on the circulation of the uvea, of which the iris is the sole portion which is clearly visible.

In man, unfortunately, the iris circulation, like the choroidal circulation, does not lend itself to these investigations, and however great was our desire, we were unable (even with the magnification of Czapski's glass) to observe any iris pulsation. It may be that we should see in this fact a confirmation of Henderson's opinion, that the arterial circle of the iris in man is in reality a venous circle.

The dog, which Leplat used for his researches, possesses in the periphery of the iris, particularly on the side toward the nose, veritable arterial trunks of which the beatings can be seen with a magnifying glass of low power or even, under good conditions of lighting, with the naked eye. The observation of the pulsatile arterial reactions is thus very easy and enables us to study, as our Belgian colleague has done so well, the pressure in the vessels of the iris. On our side, we have undertaken a series of researches for the purpose of studying comparatively the iris circulation and the retinal circulation; and we have made use of the cat. The dog which is a good subject for the vessels of the iris, appeared to us to be of no service for the study of the vessels of the retina; for its central vessels when they reach the disc are already divided into fine branches surrounded by neuroglic tissue. This disposition does not allow the beating to attain the amplitude desirable.

The retinal vessels of the cat, apart from their being disposed on the periphery and not in the center of the disc, and their ciliary origin, resemble on the contrary fairly exactly the vessels of the human retina. At the limit of the dark grey disc we see three or four arteries, each with a vein running by its side. The artificial beats which we provoke must always be sought for in the immediate circumference of the disc. They are, moreover, very easy to observe; when the appropriate pressure is applied to the eyeball, three or four small arteries can be seen beating simultaneously.

The vessels of the iris are per contra rather less easy to observe than those of the dog. In order to see them well, it is necessary to make use of Czapski's microscope, and to examine the nasal extremity of the horizontal meridian. A voluminous vein (particularly if one looks very closely at the root of the iris, before its bifurcation) will be seen lifting up the trabecular tissue; at this spot we recognize the existence of spontaneous arterial movements which pressure on the eyeball exaggerates and transforms into veritable pulsations and finally suppresses completely. If the retinal pulsations of the cat are always easy to see, it must be added that the pulsations of the vessels of the iris are sometimes less so.

The comparative study of these two pulsatile phenomena has enabled us to remark that in the eye of the cat the pressure in the arteries of the iris and of the retina is practically the same; if in the average case it has been necessary to apply some stronger pressures to study the pulsation in the iris than in the retina, this is due, no doubt, on the one hand to the greater difficulty of observation in the iris. We can give as an average in the cat for the arteries of the iris and the retina, 45—100 mm. Hg., which figures are slightly higher than those in man.

These experiments on animals have led us to recognize that the systems of blood vessels of the retina and the uvea, so similar to each other in many ways, have in their normal state an identical working pressure.

We have since had fresh confirmation of this in a work of Vossius. This author had occasion to examine with the corneal microscope a filament of an iris

in a persistent pupillary membrane. On applying dynamometric pressure to the eyeball he noted that circulation was arrested when the ocular tension reached 70 mm. Hg. We know that this figure (70 mm. Hg.) is also that which we found for the normal systolic pressure.

The study of the effects of graduated and known pressures on the eyeball has also led us to other results. We have been able to observe a constant phenomenon which can be formulated as follows:

1. From an initial tension of 15 mm., there exists always a lowering of the ocular tension after each application of the tonometer, however correctly the application be made. Example, take an eyeball of which the tension is 20 mm. Hg.; submit it for a duration of three seconds to a pressure of 30 grams. We shall find immediately after that the ophthalmotonus is no longer 20 mm. Hg., but only 15 mm.

2. This phenomenon no longer exists when the same pressures are applied to the eyeballs, possessing only a tonus of 10 to 12 mm. Hg.

3. This phenomenon is exaggerated when the pressure is applied to eyeballs in which the tension has been raised artificially in our experiments by means of subconjunctival injections of hypertonic serum.

These modifications of the ocular tonus under the influence of slight pressures applied to the eyeball had already been noted in tonometric experimentation. The remark had already been made clinically in 1911 by Polak Van Gelder. Using Schiötz's tonometer he noted that in normal individuals the repeated application of this instrument, at intervals of 3 or 5 seconds, always furnished figures inferior to the first ones. If we translate into grams the pressure exerted on the eyeball, by the tonometer loaded with these weights of 5 or 7 grams, we see that it varies from 21 to 25 grams. In Polak Van Gelder's tables, we note that the difference between the ocular tensions before and after this tonometric pressure is about 10 mm. Hg.

This fact is also quite clear when dynamometric pressures are applied to the eyeball in order to measure the arterial retinal pressure. If, when by means of a given pressure we have managed to efface the last arterial pulsation, this pressure is maintained for a few seconds, it will be seen that the arterial pulsation reappears very rapidly; to suppress it, greater pressure must be applied; if this pressure is maintained the pulsations reappear. Thus the ocular tension diminishes immediately and constantly under the influence of dynamometric pressure. This fact must be noted as it explains why beginners, who proceed by feeling their way, always find very high figures in their attempts to determine the arterial retinal pressure.

This lowering of tension under the influence of pressure is moreover very transitory; to the depression there succeeds even a slight ulterior elevation which lasts a very short time.

How can we explain this diminution of the ocular tension under the influence of ocular pressure? Evidently by the issuing of a part of its liquid contents. The supporters of the continuous stream of the aqueous humor explain it by a more active filtration of this liquid, which is driven out of the eyeball by the mechanical effect of the compression. For our part we believe that the question is not so simple. Remembering the modifications of the ocular tonus under all the influences which can act upon the circulatory system of the choroid, the fall of the ophthalmotonus after ligature of the ciliary arteries or of the carotid, after stimulation of the "sympathetic," its rise which follows inhalation of amyl nitrit and after division of the sympathetic, we remain convinced that the compression of the eyeball acts by emptying the choroidal system, and that this mechanical expansion of the external coat of the eye brings about the fall of the ocular tonus, which rises again when little by little the blood fills once more the exceedingly rich vascular system of the uvea. (See p. 777.)

Plastic Operations About the Eye.

Dr. John M. Wheeler, New York. Surgery of the eyelids, eyebrows and orbital region should be performed by

ophthalmologists, and not by those who have no special knowledge of surgery of the eye region. General surgeons are uninformed on the peculiar features and requirements here, and they are not trained in the refinements essential to success in plastic eye surgery.

Plastic eye work enriches the field of ophthalmic surgery and broadens the scope of the ophthalmologist without interfering with the special skill which he must develop in order to perform successfully the important operations on the eyeball proper. And certainly nothing should be allowed to detract from the high importance of the skill and refinement and judgment called for in handling such conditions as cataract and glaucoma.

In studying and applying methods for handling cases of deformity about the eyes, I have always had in mind these two things: First, to avoid adding new deformities thru my surgery. Second, to develop procedures which are as simple as possible, and which are appropriate for general adoption by trained eye surgeons.

In order to make satisfactory comparisons of the relative merits of different sorts of grafts in corrective surface work of the eyelids and thereabouts, I have used different types of grafts and different methods on the same patient. One patient who needed more than one correction received a true skin flap from the inner aspect of the arm, a free graft of true skin from one temple, and a pedunculated flap from the other temple. Another received epidermic grafts from the thigh, and true skin from an upper lid. Another, a pedunculated flap from the temple, a free graft from the arm and a free graft from the upper eyelid, and so on. Thus, by making different sorts of combinations, it was possible to study results in an unprejudiced and convincing way.

CICATRICIAL ECTROPION.

In most cases of traumatism resulting in ectropion, only a single eyelid is involved, and the lower lid suffers more frequently than the upper. The most satisfactory correction can be obtained by means of an inlay of true skin from one of the upper lids. It is surprising how seldom one or the other upper lid will fail to provide ample skin for grafting. A match in appearance is assured without the creation of an additional deformity. There need be no fear for the upper lid on account of removal of the skin. A fusiform flap of true skin 50 mm. long and 25 mm. wide can be taken safely from most any normal upper eyelid, and some could furnish much greater width. In one young adult I have taken, at intervals of a few months, three separate grafts from the left upper eyelid without lagophthalmos or appreciable deformity. The stretching of this skin tissue and readjustment to the normal appearance are remarkable.

A description of this operation was published in the Journal of the American Medical Association, Nov. 19, 1921.

Occasionally we have to deal with extreme ectropion of all the lids associated with facial burns, and large loss of the cilia and brows. Usually the outer canthus is pulled well down out of position and there may be epicanthal folds. In such cases it may not be feasible to implant true skin grafts, and it is convenient to resort to epidermis. Fortunately, in these bad burn cases epidermis matches the scarred tissue of the face about as well as true skin from a distant part, and it is possible to cover as large areas as necessary.

A figure shows at the lid margins the little areas denuded of epithelium in preparation for the adhesions to hold the lid margins together. These adhesions are important in the correction of all ectropion cases, but especially so in the severe cases, where large denuded areas have to be covered. After passing the sutures to secure apposition of the raw areas of the margins, a single large piece of epidermis from the outer aspect of the thigh is placed over the lids, overlapping the margins of the denuded areas all around. For this purpose a graft with an area of 9 to 12 square inches may be needed. No sutures are used for it, but the pressure of a secure dressing and bandage are relied on to hold the graft firmly in place. After placing the skin in

position, a slit should be made in it just in front of the palpebral fissure to provide for drainage of conjunctival secretion. It is well to make a slit also in the rubber tissue which is placed over the graft. Dressing and after care are the same as described for the dermic graft. In cases with bad facial scars, especially if there is a tendency to keloid formation, it may be wise to leave the interpalpebral adhesions for as long as a year, and it may be necessary to graft a second time if late contraction of facial scar tissue pulls the palpebral fissure out of place. When the work is completed, the external canthus should be on a level with the internal. In making the preparatory dissection, the tissues should not be handled roughly. Artery clamps should not be used unnecessarily and no ligatures should be used on bleeding vessels.

Removal of the true skin graft from the upper lid is very simple as compared with the dissection of a dermic flap from the arm, thigh or elsewhere. Almost no allowance need be made for contraction. In removing the graft, I do not use a grasping forceps of any kind for fear of bruising the tissue cells.

The graft should be covered with rubber tissue, having the slightest smear of sterile petrolatum. I prefer to put the tissue on in two layers with the grains running at right angles, to guard against the possibility of perforations thru separation of the tissue fibers. The rubber tissue will prevent the skin graft from getting dry. If a covering other than rubber tissue is chosen, it should be of smooth surface, pliable and without perforations. Over the tissue, gauze fluff is packed and secured firmly by adhesive plaster, and then by pressure bandage, which in turn should be secured by adhesive plaster. It is well to put a separate dressing over the other eye, to be left for two or three days, and then to be cut down without disturbing the main dressing and bandage. This should be left for six days, at the end of which time it is removed with the utmost care, and the graft painstakingly cleansed with damp (not wet) boric acid sponges, and all sutures taken out. At the first dressing the grafted skin will appear pink, not anemic like a graft from the arm. The outlines of the beautiful inlay are hardly seen even at the first dressing. Rubber tissue, gauze dressing and bandage, changed every two days, are continued for another week; then dressing is no longer necessary, and the graft is kept smeared with a little sterile petrolatum.

About three weeks after operation, it is well to start massage with petrolatum. This is kept up daily for several weeks. The adhesions between the lids should remain for at least three months. They should be left until all tendency to malposition of the lid has passed. They are then cut with the scissors and no deformity results from them.

In ectropion, I now find use for pedunculated flaps only in those cases where there has been loss of bony tissue and a consequent depression to be filled in. Here thickness is called for. A flap can be turned down from the temple or forehead by the well known methods of procedure, and the thickness can be gauged to suit the needs of the case. That is, where the depression is greatest the flap can be made thickest.

RESTORATION OF BROW.

Dislike for making new deformities and desire for simplicity have kept me from turning pedicled flaps from the hairy scalp or elsewhere in attempts to bring about the formation of new brows. I have used only free grafts. Loss of the brow is usually not complete, and rather satisfactory patch work can be done in some cases by taking from one part of the brow to fill out another, or by taking from one brow to help out its fellow.

Possibly the most satisfactory way of grafting for an entire brow or a large part of one is to remove a flap of scalp from the occipital region of the desired size and shape and place it in position. The hair follicles are usually closely placed in the middle of the occipital region, and they are well slanted. Moreover, no visible scar re-

sults. One objection to the scalp graft is that the color of the hair may not accurately match the color of the fellow brow. Another objection is that a part of the graft may be lost. The reason for this is that the scalp tissue is thick, and the graft cannot be made thin without injuring the hair follicles, which are long enough to go entirely thru the scalp skin. One cannot transplant thick detached grafts with confidence, as is possible with thin skin grafts.

In preparation for removal of the graft, the scalp area should be shaved, cleansed with alcohol and ether, and painted with iodin. The graft needed is usually rather long and pointed. The curved incisions for its removal should slant toward each other, so that a cross section of the graft is like a truncated wedge with base at the surface. This will fit deeply into the depressed exposed area in the brow region, as the sides of this depression will be "banked." In taking a scalp flap there is inevitable destruction of some of the hair follicles, but with a complete take or nearly that, a good mat of hair should result. The graft should be sutured in place and firm pressure applied. The resistance offered by the skull is a valuable help to the pressure dressing in getting firm contact between raw surfaces.

RESTORATION OF CILIA.

There are several types of cases calling for restoration of the eyelash line. I should like to give four suggestive ideas.

Occasionally in extreme ectropion the upper lid margin is found in the brow, and it is possible to make the releasing incision in the lower part of the brow instead of below it, and so carry down a hair line at the time of the dissection made in preparation for the epidermic graft. This idea is decidedly worth bearing in mind.

Where the lid margin is not actually in the brow and epidermic grafts have covered both upper and lower lids, and the lids are held together by adhesions in front of the eyeball. Hairs should be taken as largely as possible from the nasal end of the inferior border of the brow, as here they are more numerous than at the temporal end and stand out in better direction. The idea in turning the graft upside down is to bring the hair line as near the lid border as possible. A few weeks after the interpalpebral adhesions have been cut the lid margin should be trimmed off evenly close to the new cilia line.

3. In another type the eye may have been lost, and a part of the lid margin with the cilia may have been destroyed. In such a case a curved incision may be made near the injured margin, and the skin below it dissected up. Then this skin is rolled back and tied down to the fundus of the socket with silk sutures. Then a graft from the nasal end of the lower margin of the brow is placed so that the hairs will line up with the remaining cilia as accurately as possible. The hairs of the graft really overlie the unrolled skin and so fall in line with the cilia. A little trimming of the margin of the lid may have to be done later.

4. Another possibility should be borne in mind. When skin of the upper eyelid is used for ectropion of a fellow upper lid or a lower lid, it is feasible to carry the upper graft incision into the lower part of the brow and so carry with the graft a line of hairs to be placed for cilia. The graft below the cilia line may be made as large as desired. If this idea is used, in order to bring the hair line to the lid margin, the graft is carried directly to the denuded area, without turning, for the lower lid; but for ectropion of the upper lid the detached graft has to be turned upside down.

It would be absurd for one to expect a perfect line of eyelashes to result from any attempt at substitution, but patients are delighted with the imperfect results which can be obtained by the schemes suggested.

NOTCH AT LID MARGIN.

In the Archives of Ophthalmology in January, 1920, I referred to a method of handling this condition and called

the little procedure the "Halving Operation."

The little mass of scar formation around the laceration of the tarsus is excised, and the tarsal flaps are cut clear across to give accurate apposition. Assuming that the deformity is in the upper eyelid, care should be taken to excise fully as much tarsus at the upper margin as at the lower in each flap of the tarsus. This is important, and even a slight overcorrection of the notch can be obtained, at the surgeon's discretion, by diverging the tarsal incisions from the lid margin, so as to make the tarsal flaps slightly shorter above than below. The skin and orbicularis are cut so as to make a flap on one side, and a rectangular area of tarsus is exposed on the other. If necessary, to prevent tension on the wound, especially if the eyeball has not been removed, a canthotomy should be performed, and the breach of the external canthal ligament should be severed to release the tarsus. The little tongue of skin is trimmed enough for adjustment, and a small triangular piece of skin may be removed above to avoid puckering. The conjunctiva and tarsal flaps are first sutured. A mattress suture is carried thru the flaps, and tied after passing thru a small rubber plate cut from sterile tubing. Sutures are introduced to give good apposition of the skin flaps. The mattress suture should be removed in two or three days, and the others about five days after operation.

An important point is that tarsal wound and skin wounds should never be in the same position, but should be placed in such a way that there is overlapping. Thus, what is known in carpentry as halving is accomplished, and union is assured, even if there is a little sloughing of the skin. Furthermore, recurrence of the notch formation is prevented. After healing there is no break in the continuity of the lid margin or in the row of eyelashes. In handling notch cases, different short-cut procedures have been tried, but the "halving" method has been the only satisfactory one in my hands.

TRAUMATIC COLOBOMA OF LOWER LID NEAR INNER CANTHUS.

A rather common injury is that of laceration of the lower eyelid thru the canaliculus, extending downward and outward more or less accurately along the lower orbital margin. Improper primary healing of such a wound results in a coloboma near the inner canthus and ectropion of the lid near the coloboma. Complete correction of this deformity can be obtained by a simple maneuver. Scar tissue at and near the coloboma is dissected away. An incision is carried from the coloboma downward and outward. The attachment of the lower lid to the external canthal ligament is severed, and if necessary, another incision is carried outward and slightly upward from the outer canthus. The flap is undermined as much as necessary, so that its apex can easily be carried to the inner canthus. The amount of dissection necessary will depend upon the amount of tissue loss at the coloboma. Sutures are placed diagonally along the incisions in such a way as to advance the whole flap and have as little pull as possible for the sutures at the apex of the flap during the healing process. Of greatest importance is the adjustment of the tissues at the apex of the flap. The small amount of skin just external to the inner canthus is carefully and completely undermined. The skin and orbicularis are stripped from the anterior surface of the tarsus so as to expose 4 or 5 mm. of the tarsus. A suture is carried thru the exposed tarsus, and then it is carried well into the internal lateral ligament or into the deep fascial tissue behind it, so as to get a firm hold. A mattress suture mounted with two needles is carried thru the exposed tarsus, then thru the flap of the skin which has been dissected up near the inner canthus. When the sutures are snugly tied the apposition will be secure, and the lid will lie well back against the globe and well up to the proper level, or even high enough to be in a position of overcorrection. Without this careful adjustment and definite overlapping of

raw surfaces one cannot be sure of complete obliteration of the deformity.

RESTORATION OF OBLITERATED SOCKET.

Many suggestions have been made for making new sockets after obliteration, and many disappointments have resulted from various sorts of procedures. The method which I have adopted I can recommend with confidence. Epidermis has been chosen as the most satisfactory tissue for lining the cavity, as it will give a thin, pliable lining, free from hairs and an excess of oily secretion. The lateral aspect of the thigh is usually the most convenient place from which to take it. No elaborate preparation is necessary. The part from which the epidermis is taken is shaved and cleansed with alcohol and ether. Then with a long bladed knife or razor with a keen edge, a large piece of epidermis is taken. For taking the graft I am now using the Stille (Norwegian) graft knife, and an interested barber gives it an exquisitely sharp edge, without which it is impossible to get a large graft free from perforations and free from true skin.

If the lid margins are adherent, they are separated by an incision. The dissection is then carried out in such a way as to separate the lids from the orbital contents. The following points should be borne in mind:

1. The dissection must be kept superficial, so that in front of the dissecting knife or scissors there is only lid tissue, i. e., only skin, orbicularis, the thin fascia of the lid, and the tarsus. It is not necessary to save the tarso-orbital fascia with the lid. Carrying the dissection back into the orbital tissue is probably one of the most common causes of failure.

2. Not only the superficial plane of the dissection, but the extent and limitation for the dissection are of importance. Temporally and below, the dissection should be carried well to the orbital margin or even 1 mm. or 2 mm. beyond it, as the graft must adhere to the periosteum of the anterior aspect of the orbital margin. On the nasal side, the dissection should be extended to the anterior crest of the lacrimal groove and to the orbital margin above it. In dissecting at the inner canthus the caruncle should be saved if it has not been destroyed. The graft will adhere to the posterior surface of the caruncle and give it a permanent lining. In the division of the tissues above, the dissection should be carried behind the orbital rim but not necessarily to the roof of the orbit. In some cases the levator can be saved.

3. In preparing the bed for the graft all cicatricial and granulation tissue should be removed. Excision of granulation tissue is especially important as the contraction of such tissue may result in contraction of the socket. Reduction in size of the newly made socket is due to contraction of the underlying tissue rather than to contraction of the skin itself.

4. A common fault which manifests itself after restoration of the socket is that of too great thickness of the lid margin. This deformity can be obviated by cutting away the tarsus. If the skin graft must extend completely to the margin of the eyelid, the tarsus may be split and thinned. If the graft is to extend nearly to the margin, sufficient tarsal plate may be cut away so that the graft will set in as an inlay. In any case, enough tarsus should be left to support the cilia, but not necessarily any more. A form should then be moulded to fit the cavity. Dental impression compound is ideal for this purpose. Ordinarily for full restoration of the socket, the dimensions are approximately as follows: Length 40 mm. to 45 mm., width 30 mm., thickness 5 mm.

The ideal graft for socket restoration is one without perforations which is made up of epidermis only, free from layers of true skin, and large enough to be wrapped around the form of impression compound with generous overlapping. This means a graft 3 1/2 to 4 inches long and about 3 inches wide.

The graft of epidermis is immediately wrapped about the form of impression compound, raw surface outward. and overlapping on the surface which is to be anterior. The form, completely

covered with epidermis, is forced into the socket cavity. It is not necessary to remove small blood clots before placing it as they will not prevent a take. The overlapping portion of the graft is placed forward so that if the edges are disturbed by manipulation they can be carefully replaced thru the palpebral fissure so that every part of the form will be covered.

No sutures are used. A pressure bandage is applied, and over this adhesive strips. Very firm pressure is of importance to secure accurate contact at all points and to keep the cavity absolutely obliterated. This first dressing is left in place for a week. The form is left in place and is not touched for about three weeks. It is then removed and left out permanently. The artificial eye may be introduced at any time.

If the surgeon has been successful in carrying out the technic, the result will be a permanent socket, extending well beyond the canthi and of sufficient dimensions all around. The lids will be normally thin and pliable, and the thin walled socket will not prohibit motility of the stump and artificial eye, altho the movements of the eye will be somewhat less than those following ordinary enucleation.

An interesting development is that usually lacrimal secretion will find its way into the new socket and maintain a comfortable moisture. As far as I know, trouble never results from activity of the lacrimal gland.

Blepharoplasty for Bilateral Lagophthalmos.

Dr. J. N. Roy, Montreal, Canada, reported the case of a nun, who came for double lagophthalmos existing for a quarter of a century. At the age of twelve years, while working in a mechanical laundry, her hair caught in a belt; her head was drawn against a joist and she was instantly scalped. Her eyebrows were torn away and lower lids were drawn up covering the palpebral opening. When temporofrontal growth of epidermis occurred, the external canthi were raised even with the upper lids, and after a year the patient could no longer close her eyes. A double lagophthalmic keratitis appeared, and the skull cicatrized very slowly.

Roy made, under local anesthesia, an incision of about 5 cm. in the region of the canthus and external quarter of the displaced eyebrow. After dissection and drawing down of that part, he took from the cheek an appropriate flap to fill up the space so produced. This flap was kept in place by a silk suture, and after detachment of the skin adjacent to the incision in the neck, the lips of the wound were well drawn together and sutured.

This blepharoplasty, practiced on both sides, gave a perfect result as to opening and closing the eyes, even as to the symmetry and horizontal position of the palpebral openings. Photographs illustrated the condition.

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CAUSES OF OCULAR PALSIES.

The paralysis of one or more ocular muscles, producing dizziness, nausea, double vision and manifest inability to turn the eyes in certain directions, should at once raise the question of causation. Clear understanding of the pathologic condition present, light upon its exact location, prognosis as to its probable course and outcome, and especially the early treatment of the condition, rest upon what is known of the cause.

Trauma may be thought of first, and should be carefully inquired for. The motor nerves that supply the extraocular muscles run a course that exposes them to injury from falls or blows striking on the head, and the later consequences of such trauma. Lacerations of the nerve tissue or sheaths, pressure on the nerve fibers by resulting exudate or hemorrhage, are likely to occur. It is possible that violent shaking may lead to functional impairment, or subsequent degeneration in the neurons of the nuclei of the nerves.

The double vision and other symptoms may not appear immediately after the injury, so that their dependence on it may not be thought of. Inflammatory exudate and hemorrhage may produce their effects slowly, and failure of nerve function may be gradually progressive after pressure has arisen. The diplopia may not be fully established for many days; and earlier symptoms ascribed to the general effects of the shaking up, rather than to the embarrassment of ocular movement. Closure of one eye, or swelling of the lids, or confinement to bed for other effects of the accident, may delay the discovery of the diplopia. Injury to other parts of the nervous system, concussion and mental hebetude, even when not very manifest, may prevent the patient from recognizing the character of his disturbance of vision.

Next to trauma may be mentioned nontraumatic lesions of various sorts situated mostly in the orbit. These are unusual, but on that account more obscure and liable to be overlooked. They include inflammations involving the muscles, their tendons and sheaths, or the motor nerves supplying them; or

of the periosteum and other connective tissue structures of the orbit. Such inflammations arise in connection with general disease, or by extension from the bony walls of the orbit or the neighboring sinuses. Malignant disease invading the orbit, especially by metastasis, may cause paralytic disturbance of the ocular movements, the origin of which is likely to be very obscure. Otitis media causing abducens paralysis might be mentioned with such local lesions. From these local causes the paralysis may arise by nerve block thru pressure, or by inhibition to avoid disturbance of inflamed tissue by movement.

The share of lues in causing ocular palsies has probably been more justly estimated in the past, 20 to 50 per cent, than the proportion of luetic inflammations of uveal tract. Yet new statistics, based on the application of new methods of diagnosis, and more rigid logic in dealing with the grouping of such cases, are needed. These questions all arise: Is lues present? If present, does it cause the paralysis? It is erroneous to assume because the patient has lues, it is certainly the cause of his ocular palsy. Thirdly: What is the relation of parasyphilitic disease to syphilis? Then syphilis causes so many different kinds of lesions. Is the paralysis due to a gumma, a vascular lesion, or a toxic degeneration of nerve cells? Prognosis and treatment will depend on the answering of such questions by complete diagnosis.

The relation of focal infections to ocular palsies has not been studied much beyond the observation of suggestive coincidences. Focal infections fall heir to the cases formerly classed as rheumatic, with all the indefiniteness and uncertainty that pertained to the real nature of such cases in the past. Weakness of accommodation associated with focal infections has been more frequently reported. As to the manner in which focal infections cause paralysis of the eye muscles, the old explanation of neuritis seems to hold good and receives support from the experimental studies of the tendency of certain strains of streptococci to produce neuritis.

Of acute infectious diseases that cause ocular palsies, diphtheria leads, with influenza and epidemic encephalitis closely following. In the latter disease, double vision is often a first symptom, and may be transient or intermittent. With diphtheria, influenza and most other acute infections, the paralysis of the ocular muscles is a sequel. With diphtheria it may be first noticed several weeks after convalescence from the general symptoms and local lesions in the throat; and is usually a much later effect of the diphtheria toxin than paralysis of accommodation.

The share of such chronic diseases as tuberculosis, diabetes, gout, etc., in causing paralysis of ocular muscles is probably due to their tendency to cause local lesions in various structures, or to lower the plane of vital processes in a way that might be compared with the tendency of old age. Such lessening of resistance must be taken into account in connection with all influences that tend to cause ocular palsies, and should not be accepted to the neglect of careful search for more active causes. Among active causes, poisons must not be forgotten. Lead, carbon monoxid, botulism, alcoholism, and ptomain poisoning are the more important poisons which damage the nuclei of the ocular motor nerves. Abducens paralysis may attend spinal anesthesia by cocain, or procain.

Recurring oculomotor paralysis is a rare but very interesting condition. It begins with intense headache, commonly unilateral, and often there is nausea, vomiting, fever and general weakness. The paralysis due to the first attack commonly recovers completely. But recurrences leave progressively greater paresis, until paralysis may be permanent and complete. Whether it is caused by some toxin or by repeated infective or anaphylactic conditions is still unknown.

E. J.

TAKING THE NEAR POINT.

The power and range of accommodation of an eye are known by measuring the refraction and taking the near point. The refraction is measured, as a matter of routine, in a large proportion of cases; but the taking of the near point of distinct vision is often neglected. Yet asthenopia arising from eyestrain usually depends on strain of accommodation. It is generally assumed that if hyperopia and astigmia are present, the accommodation will be strained by much near work, and the assumption is mostly correct. Correction of the error of refraction may give relief without any study of the power of accommodation in the particular case. But one cannot know how much the accommodation is strained, without knowing how much of it there is to do the required work.

The best advice is based on complete knowledge of the case. It is a neglect of professional duty to prescribe for strain of accommodation on an assumption based on age and general symptoms; when we can know so easily just what the accommodation is and how much can be properly expected of it. The data regarding it are usually all at hand, except the position of the near point, and this can be so quickly taken by simple methods, that the neglect of it is inexcusable.

It is not only in presbyopes that the near point should be taken to determine the convex lenses to be given for near work. Weakness of accommodation sometimes exists and causes annoying and persistent asthenopia long before the "presbyopic age." Diminished but still active accommodation may continue to cause eyestrain, much later than the common assumption would have it eliminated. Squint in children and heterophoria in older patents, may be closely dependent on defective accommodation. The state of the accommodation can supply an excellent indication of the strength and endurance of the general neuromuscular system. Knowledge of the accommodation supplements what we learn by taking the visual acuity and state of refraction, giving certainty and definiteness to certain indications they furnish. The taking of the near point should be as much a part of the routine examination as use of the distance test letters and trial lenses.

The test cards commonly used for determining the near point have been better for roughly testing the near vision of presbyopes, when the near point has moved off until distinct vision at the working distance is not possible without the help of convex lenses. The finest type in the Jaeger series is clearly visible at one half meter to eyes with standard vision; and is therefore quite unsuited to determining the near point at shorter distances. To attempt to determine the near point at one-fourth meter, where it commonly is when presbyopia begins, with Jaeger No. 1, is about like trying to measure the visual acuity at twenty feet by use of forty feet test letters. Type of ordinary type metal cannot be cast fine enough for taking the near point of young patients. The molten metal is not sufficiently fluid to run into such fine lines, and leave such small spaces between them. Test cards for taking the near point must be obtained by photography, reducing the printed matter produced from larger clear type to the size required for this purpose.

Various optical houses have put out near vision test type produced in this way, the best having a type just legible for standard vision at one-fourth of a meter, ten inches. But even such type does not afford a strictly accurate test for the near point of distinct vision in young persons, who have a near point of one-tenth meter or less. Reproductions of printed pages are sometimes available, that furnish letters only recognizable at the near point of children, but generally the original type has not been perfect, the reproduction defective and the size of the letters reproduced not planned for this particular purpose. Duane has rendered a real service in devising a more delicate test; altho it is not capable of the

pseudoobjective character, given to such tests, by requiring the patient to read the letters that he claims he can see clearly.

But the fact that the tests at our disposal are far from ideal, is no excuse for not applying more generally the tests that are readily available; or that can be improvised anywhere that the fine print used in the advertising pages of most magazines can be found. In early life the position of the near point gives a quick, simple indication that the eye tested is hyperopic or myopic. This is true even of eyes with lowered acuteness of vision; if the size of type made out at the near point is compared with its distance from the eye and the visual acuity, as tested on the large test type at five or six meters. In early adult life the position of the near point still indicates the probable kind and amount of ametropia, or is evidence of paresis of the accommodation or early presbyopia. For the period of presbyopia, it is the key to any satisfactory choice of lenses. At all ages it should be considered in relation to convergence and esophoria and exophoria.

The actual taking of the near point is an extremely simple matter and consumes very little time. Hold the test type used in front of the eyes far enough away to be easily read. Then slowly bring it closer to the eyes until the reading ceases in spite of the patient being urged to make the greatest effort to keep the print clear. A graduated rule or scale held in the other hand gives the distance of the card from the eye; or, as Duane prefers, from the anterior focus of the eye, 14 mm. from the cornea, where the spectacle glass would usually be placed. Holding the card in this position, alternately cover one eye and then the other and let the patient state whether it is equally clear to both. If not, the near point should be taken for each eye separately. All this can be done in a minute or two, and no other part of the routine examination will yield more for the time occupied by it. E. J.

BOOK NOTICES.

Diseases of the Eye. M. Stephen Mayou, Surgeon to the Central London Ophthalmic Hospital. Third edition, 12 mo., 334 pages, 145 illustrations, 4 color plates. London and New York, Oxford University Press.

This book is written for students who are beginning their study of the subject, with an appreciation of the fact that the medical curriculum for the medical student is already overcrowded; and for the practitioner, who wants to find quickly something about conditions that he does not keep constantly in mind, so that he turns naturally to the one book about such diseases with which he has become familiar.

It admirably fulfills its purpose, presenting the most important facts, stated briefly and clearly. The reduction of an account of ophthalmology to meet the needs of the student is not merely a shortening of each chapter, a percentage allotment of space devoted to each branch of the subject as compared with that given to it in a complete textbook; certain things have to be omitted entirely, while others need to be treated rather fully. This requirement has been met by Mayou with wise discrimination.

There is a chapter on methods of examination, 23 pages, which the student should master thoroly if what he learns in other parts of the book is to become available for the service of patients. In this chapter, one may be surprised to find five pages devoted to examination of the field of vision with illustrations of Mayou's and Elliot's scotometers, and McHardy's perimeter. But a little reflection shows that the field of vision has a diagnostic importance in general medicine and surgery, that has not been appreciated; and we must expect that the interest in it on the part of general practitioners will be greater than has been manifested in the past.

The second chapter, on elementary optics and refraction, 31 pages with 15 illustrations and diagrams of meri-

dians of astigmatism and cylinder axes, gives sound practical instruction, carried as far as is possible in such limited space. It is calculated to enlist the interest of the student, and make him believe, if circumstances place him in a community where better service in dealing with errors of refraction is not available, that he can correct ametropia as well as he can treat disease, when he leaves the medical school to begin his real life training in his chosen vocation.

Diseases of the conjunctiva we would expect to have extended consideration in a book intended for the education of general practitioners. It has a chapter of 48 pages, containing 22 illustrations, 13 of which deal with the pathologic histology and bacteriology of the conjunctiva. Cornea and sclera have a chapter of 23 pages, diseases of the uveal tract have 28 pages, the lens 11 pages. The retina, vitreous and optic nerve are considered in a single chapter of 40 pages. Glaucoma has 11 pages, the extraocular muscles and movements of the eye 14 pages, diseases of the lids and lacrimal apparatus 18 pages, and the orbit 7 pages. There is a separate chapter on operations, containing 44 illustrations, among which occur these errors; Fig. ures 117 and 118 are transposed, and in Figure 139 the positions of the two parts A and B are reversed.

The appendix contains a series of 26 prescription formulas, of most general usefulness, and a statement of vision required in the dozen different branches of public service. There is a good index, 10 pages. The color plates represent 2 normal and 6 pathologic conditions of the ocular fundus. The volume is excellently printed and very convenient in form. We predict that as it comes to be better known in America, it will be rather widely used among medical students.

E. J.

Myopia and Its Treatment by Tuberculin. George Hirsch, Halberstadt, Germany. 49 pages.

This is a paper covered brochure of 49 pp., devoted to the contention that myopia is a condition of the eye caused by weakening of its coats, due to changes in their nutrition caused by tubercular toxins; and to the treatment of the condition by the injection of doses of tuberculin, and also to the prevention of myopia by prophylactic dosing with this remedy. A revised English edition is in preparation—subscription at the author's address—the latter we hope in a little better English than the advertisement.

The contentions of the author are based upon the relative frequency of myopia and tubercular conditions, presumably in his own country; as well as upon the researches of other authors which show some relation between tuberculosis, the disordered general and local nutrition, and shortsightedness.

All children are hyperopic when born. Progressive myopia begins from the 5th to the 7th year, when the eyes are not used so much for close work as later on. He also shows that most well read people are not myopic, and that most of the myopes go to the optician and not to the eye doctor. He quotes Koch, Saemisch, Bertrams and many other authors in regard to the formation of antitoxins in the blood. These substances fight off most of the diseases of men. Tubercle bacilli are found in the cornea, conjunctiva, etc., and some diseases of the eye are accepted as being of tubercular nature.

He shows that myopia is due to the formation of posterior staphyloma, and is produced by changes in the nutrition of the posterior portion of the eye, which is supplied by the short ciliary arteries, which supply the choroid. Many authors are quoted showing the relation between myopia and civilization, and as to myopia, syphilis and tuberculosis. Statistics are given regarding the relation of these diseases, the average being 10 to 30 per cent of myopes. According to the author the tubercular toxin has a special action upon the posterior portion of the eye. Cases are quoted. The tubercular toxin also produces an effect upon the anterior ciliary arteries and in the production of squint.

Tubercular diseases of the eye and of the ear are sometimes combined. Cases are quoted of myopia and tubercular diseases of childhood. A number of pages are given with case histories of myopia in the family. He thinks that the tubercular toxin often selects the one eye without affecting its fellow. Cases are treated by him with doses of antitoxin seemingly causing betterment, and he apparently believes that protection is afforded the child by prophylactic treatment by tuberculin.

H. V. W.

Essentials of Laboratory Diagnosis. Designed for Students and Practitioners. **Francis Ashley Faught,** M. D., Philadelphia. Seventh edition. 78 illustrations, 11 plates. Philadelphia. F. A. Davis Co.

This is a very readable book, being written in a manner that the general practitioner, specialist, and the student may make a complete review of the progress of laboratory diagnosis to date. Even tho he sends all his specimens and laboratory cases to the laboratory worker for such examination, if he would read this book and then keep it for reference in special cases, he would himself, as well as his patients, gain by its possession. It does not take the place of exhaustive text books, but supplements them by descriptions of simple and reliable methods of obtaining the results of laboratory examinations. The newer methods are well described, the chemicals discussed, the microscope and its appurtenances, clinical hematology, the spectroscope, newer methods of blood examination, sphygmomanometry, animal parasites, feces, urine, blood, fundus, milk, etc., and a full appendix, giving laboratory test stains, standard solutions, etc., with a full glossary.

The reviewer found of special interest the discussion of blood pressure and the use of the sphygmomanometer to determine it, the description of sero diagnosis as to syphilis, typhoid and other diseases. The book is well printed and sufficiently illustrated.

H. V. W.

CORRESPONDENCE.

Asteroid Hyalitis.

To the Editor: The paper of Bachstez, "Acid fat lime as basis of a species of scintillatio corporis vitrei," abstracted in the JOURNAL (Vol. 5, page 414), demands some comment which will clarify facts and thus prevent confusion later on. The description given by Bachstez of the condition, is so characteristic as to be identified readily with that of asteroid hyalitis by anyone familiar with this clinical entity created by Benson in 1894. (Trans. Ophth. Soc. U. K., v. XIV, p. 101.) Bachstez himself seems to have felt that his cases belonged in another group than scintillatio and to have intended to express this by the words ". . . a species of. . . ."

In spite of the many cases recorded in the American literature (Holloway, Stark) and the comparative frequency of the symptom complex—I have notes of about eight cases of this kind and none of *true* scintillatio—this confusion can be explained by the fact that Benson's description has found notice in the textbooks only very lately, that of Weeks in 1910 being to my knowledge the first to mention it; even the latest German edition of Fuchs' book by Salzmann does not separate asteroid hyalitis from scintillatio corporis vitrei.

Whether asteroid "hyalitis" is the proper name for the condition is still a question, but until a better one is found, Bachstez' cases ought to be recorded where they properly belong, under asteroid hyalitis. This is of especial importance since they help establish the fundamental fact concerning the chemical composition of the opacities in asteroid hyalitis, announced a few months before by Verhoeff (A. J. O., vol. 4, page 155).

M. FEINGOLD.

New Orleans, La.

Radioactive Photometers.

To the Editor: In January, 1919, the AMERICAN JOURNAL OF OPHTHALMOLOGY published an article of mine, "The Determination of the Minimum Light Sense and Retinal Dark Adaptation

with the Presentation of a New Type of Photometer."

A radioactive substance "Marvelite" was used as the source of light in the photometer described, and observations made with this instrument, tho elementary in character, certainly confirmed several physiologic principles in the dark adaptation of the retina.

At the time my experimental instrument was constructed, the manufacturers of "Marvelite" were of the opinion that after a certain initial period of decrease in intensity, the luminosity of the substance would maintain a constant value for an indefinite period, and therefore, it seemed to offer an ideal test object for photometric examination, if its intensity could be standardized, and this seemed possible.

Circumstances compelled me to postpone any further investigations with the instrument for a year or more; and when I resumed work with it, I was surprised to find that the luminous discs had decreased markedly in intensity, altho they had not been mounted in the instrument until they had been aged over a period which was supposed to give them a constant light value.

I wrote to the Bureau of Standards at Washington for information concerning these luminous radium compounds, and the following letter explains why it is impossible to construct a satisfactory photometer of the type with which my experiments were conducted.

DEPARTMENT OF COMMERCE.
Bureau of Standards.
Washington, D. C., June 24, 1922.

Dr. Jesse W. Downey, Jr.,
529 N. Charles St.,
Baltimore, Md.

Subject: Use of Radium Compounds with Photometer.

Dear Sir:

1. Referring to your letter of June 21 regarding the permanency of self luminous radium compounds, and their possible use as a standard source of light in a photometer, we regret to say that none of these products are sufficiently permanent for the purpose.

2. Because of the pressure of other work, our investigations on self luminous materials have been carried on only in a very fragmentary way, and we have not recently issued any publications on the subject. However, we can say with considerable certainty that none of these materials can be expected to maintain their luminosity for a very long time. In the case of materials used for the illumination of clock faces, instrument dials, and similar purposes, the useful life is ordinarily two to five years, even if the materials are made up of genuine radium. The reason for this relatively rapid decrease seems to be that the material which is made to glow by impact with radium particles becomes exhausted.

3. If you are interested in the details of investigations on the performance of these compounds, we can probably refer you to some publications covering researches made elsewhere.

Respectfully,
F. C. BROWN,
For the Director.

As my paper was abstracted and favorably commented on in several journals and at least one text book, namely, The Year Book of Ophthalmology, The British Journal of Ophthalmology, and Elliot's, A Treatise On Glaucoma (Second Edition), I feel that this explanation of my failure to produce a practical photometer warrants publication.

JESSE W. DOWNEY, JR.

Baltimore, Md.

Molluscum Contagiosum, Errata.

To the Editor: My father, some time ago, asked me to call your attention to some errors in the abstract on Molluscum Contagiosum, which appeared in the digest of literature, O. L., for March, 1922, page 145. He thought it might be possible to make a correction in some future issue, of the whole last sentence of the abstract, which should have read "they conclude:—that molluscum contagiosum may exist on the skin of the lids is well known; that it may cause conjunctivitis is not well known to ophthalmologists; that a single molluscum nodule on or near the

lid margin, even without the distinctive umbilication" and so on as it is printed.

The errors, while only involving about three words, have entirely changed the sense of the conclusions, and he would appreciate it if they were corrected. I also wish to correct a report from one of our local society meetings, appearing in the AMERICAN JOURNAL OF OPHTHALMOLOGY, 1922, No. 7, page 567. In speaking of the use of the thermophore, the abstract states, "Dr. Patton reported, that he used 150° centigrade" which should, of course, be Fahrenheit.

Sincerely yours,
S. R. GIFFORD.

Stops Unauthorized Statement.

To the Editor: I am enclosing a letter I have just written to the Umbsen Manufacturing Corporation, which will speak for itself, as I feel sure there are a number of men who would wish to take the same action I have done, but may now find it unnecessary. I am sending you the letter, which I wish you would publish in the AMERICAN JOURNAL OF OPHTHALMOLOGY.

July 28, 1922.

Umbsen Manufacturing Corporation,
Rochester, N. Y.

Gentlemen:

A publication issued by the Umbsen Manufacturing Corporation entitled "The Verdict of the Ophthalmological and Medical Professions on the Hydro Eye Spray," Bulletin No. 2, has just come to my attention. The following quotation appears therein:

"Dr. J. W. Burke, Washington. Approves of spray and says it will have hygienic value. Cannot see where it will be of great use in professional treatments. Says—'Take this to the people and they will approve. The profession cannot dispute its claims to merit.'"

I have never made any such statement to any member of your company, nor have I authorized any such statement to be made, or my name used for advertising purposes.

I must therefore insist that you *immediately* discontinue the circulation of this or any other bulletin containing the statement above quoted. I must have a reply to this letter within a week assuring me of the discontinuance of this bulletin, otherwise I shall have to bring it to the attention of the United States postal authorities and place the matter in the hands of my attorneys to take such action as they may advise.

Very truly yours,
JNO. W. BURKE.

Washington, D. C.

BIOGRAPHIC NOTICES.

PIETRO BAIARDI was born in 1862. He finished his medical course at Turin, and took up the study of Ophthalmology in Reymond's clinic. Later he was Professor at Genoa, and in 1911 was called to succeed Reymond at Turin. He contributed numerous writings on different subjects of Ophthalmology. Much of his early work dealt with problems of Physiologic Optics, including some important work on the axis in astigmatism. In considering cataract operations with reference to the postoperative astigmatism produced, he decided that the most advantageous method was the linear extraction of von Graefe. In experimental pathology he was among the first to successfully transmit trachoma from man to the apes. In the field of therapeutics he demonstrated that mercury could be recovered from the intraocular fluids after local applications.

His work on the microscopic examination of the conjunctival vessels during life, was an early forerunner of the recent work with the slit lamp. He showed minute changes of these vessels in diabetes, arteriosclerosis and nephritis.

He was a skilful operator and made important contributions to operative technic. He was the first to propose peripheral iridotomy in cataract operations. The method which he finally decided upon as ideal, however, was his subconjunctival method of extraction.

For some time before his death he had been incapacitated from active work by the disease to which he finally succumbed.

G. LODATO, tr. by S. R. G.

JOHN HERBERT CLAIBORNE, the New York ophthalmologist, died in New York City on May 27, of heart disease. He was born at Louisburg, N. C., June 29, 1861, son of John Herbert Claiborne, M.D., and Sara Joseph Alston Claiborne. His medical degree was received at the University of Virginia, in 1883. He at no time practiced general medicine, but, settling in New York City, began at once to treat diseases of the eye. He became ophthalmologist to the Flushing Hospital and Dispensary, assistant surgeon to the New Amsterdam Eye and Ear Hospital, clinical instructor in surgery, ophthalmic department of the Cornell University Medical College, and assistant to the chair of ophthalmology in Columbia University. He was a member of the New York Ophthalmological Society, the American Academy of Ophthalmology and Oto-Laryngology, and the American Ophthalmological So-

Daniel Buttrick Smith, 1840-1922
(See p. 844)

ciety. He was line captain, 12th N. Y. Vols., in the Spanish-American war. He wrote a good deal, perhaps his best known productions being "Theory and Practice of the Ophthalmoscope" and "The Functional Examination of the Eye."

Dr. Claiborne was a man of medium height and weight, of dark complexion, smooth-faced, and with very dark brown eyes and black hair. He was courteous and affable. Dr. Claiborne married, April 16, 1901, his cousin, Marie Louise Claiborne, of New Orleans, La., who survived him. By her he had one child, John Herbert Claiborne, Jr., now a student at Lafayette College. T. H. S.

ALBERTO DEL MONTE, after having spent much time in the study of histology and pathology under Palladino and Armanni, took up ophthalmology under de Vincentiis, in the Naples Clinic, where he continued to work until his death. Besides an active life as a clinician, he made numerous contributions to the literature of ophthalmology. His first writings were on pathologic and bacteriologic subjects; for example, The Biology of the Xerosis Bacillus, Petrifying Conjunctivitis, The Study of Amyloid Degeneration of the Lids and Conjunctiva. Other later works concerned black cataract, chancroid of the conjunctiva, ocular complications of variola; and a very complete work on chalazion, some results of which he intended to present at the Washington Congress. The last years of his life saw the completion of some important work along more general lines on the biology and physiology of the blood. He was an active collaborator in the Archivio di Ottalmologia.

SGROSSO, tr. by S. R. G.

DANIEL BUTTRICK SMITH, a well known teacher of ophthalmology at several medical schools, died on May 19, at his home, 1793 Coventry Road, Cleveland Heights, Ohio, at the age of 82 years. He was in practice until six weeks before his death, thereby completing fifty-five years of active work in his profession.

He was born at Middlebury, near Akron, O., April 30, 1840. When he was fourteen years of age his family moved to Oberlin. After being graduated from college there in 1860, he became principal of a school at Wabash, Ind. His medical degree was received at the Charity Hospital Medical College, Cleveland, in 1867. For a time he studied the eye, ear, throat and skin at Berlin and Vienna.

Returning to Cleveland, he practiced general medicine from 1867 to 1880, for the first few years of this time in association with Dr. G. C. E. Weber. During nearly the whole period of his general practice, he was personal physician to Amasa Stone, founder of Adelbert College of Western Reserve University. He was for a long time ophthalmic surgeon to the Big Four Railroad. For fifty years he was on the staffs of the Lakeside, Charity, City, Cleveland General and St. Luke's Hospitals. During forty-five years he taught at medical colleges, including the Charity Hospital Medical School, the Cleveland College of Physicians and Surgeons, and the Western Reserve School. It is said that he aided in the education of more than 10,000 doctors. Tho he taught so long, he wrote little.

Dr. Smith was a man of large, athletic build, inclined to stoutness, of florid complexion, and with light brown eyes and white hair. His customary facial expression was one of keen, quick observation, kindly good humor, and a strong will. He was a collector of books, coins and postage stamps, and an ardent lover of horses. He was often a member of the Cleveland Board of Education, and was once its president. He married, Nov. 17, 1868, Miss Jeanette Chiday, who died in 1914. Dr. Smith leaves no kin. T. H. S.

ABSTRACTS

Mazzei, A. Milk in Ocular Therapy. Arch. di Ott., 1921, v. 28, p. 131–154.

The author reviews briefly the literature on the use of milk in ophthalmolbgy. It was first used by Müller and Thanner at Vienna in 1916, the injections being given intravenously and subcutaneously. Darier began to use it soon after this and believed its effect to be due to stimulation of leucocytosis and phagocytosis. A number of very favorable results in all kinds of inflammatory conditions of the eye have been reported. One unfavorable report is that of Berneaud who, after 905 milk injections, reported little good effect in cases of interstitial keratitis, phlyctenular keratitis, trachoma, iritis, or choroiditis.

The use of milk injection as a prophylactic measure against infection after perforating injuries or before operation has been reported favorably by Van Lint, Weekers and Bolone. Experimental evidence in support of its effectiveness was given by Müller in 1918, who showed that it produced a definitely stimulating effect on bone marrow, and leucocytosis in animals. The author used fresh cow's milk which had been boiled four minutes. He injected about 4/10 to 1 c.c. subconjunctivally, repeating this as soon as the milk was seen to have absorbed, usually after two or three days.

He reports 16 cases of phlyctenular conjunctivitis and keratitis treated by this method, in which the only other treatment was hot applications. These cases all cleared up after two or three injections in the course of 2 to 7 days, without complications. In three cases of interstitial keratitis, he found that milk injections had no effect. In six cases of trachoma with pannus, the infiltrations cleared up and the pannus cleared up almost entirely in the course of from one to three months. The symptoms of pain were much relieved from the first. There was no effect on the lesions of the lids. In his cases, the favorable effect was seen to be proportional to the amount of local reaction obtained.

S. R. G.

Niosi, F. Cystic Lymphangioma of the Orbit, Arch. di. Ottal., 1921, v. 28, p. 219–246.

The author's patient was a woman of sixty. Fourteen years before she had noticed a tumor, the size of a pinhead, at the internal angle of her right upper lid. Operation was refused at this time, and the tumor gradually increased, producing exophthalmos. Antispecific treatment was given several times with temporary improvement. The tumor remained stationary for some years and gradually increased again. At her last appearance, her Wassermann was negative and she presented no other evidence of tumor elsewhere.

At this time, the orbital tumor was of immense size; the eye was pushed down and out as far as the junction of the lower and middle third of the nose, and the tumor had extended up and out on the brow. The visible part of it was the size of an orange. It was smooth and compressible. There was no pain. Vision was bare light perception, and movements of the eye were almost abolished. Sixty-five c.c. of fluid was removed from the tumor with a needle, and proved to be full of fat globules, degenerated cells and cholesterin crystals. The growth filled up again rapidly and extirpation under local anesthetic was done, the whole mass being dissected out in toto. It proved to be entirely outside the muscle cone and the muscles were left intact. It extended to the apex of the orbit. Healing was normal. The exophthalmos almost entirely disappeared and good motility was restored, altho there was naturally no effect on the vision.

A careful pathologic examination was made and disclosed a number of *cysts* of variable sizes with a connective tissue wall 2–8 millimeters thick. The stroma, including the cyst walls, was made up of connective tissue with some elastic fibers, deposits of calcium, and a few areas showing true bone formation. The characteristic feature was the cysts, which were lined with endothelial cells and which showed in

their walls numerous nodules of actively proliferating lymphoid tissue. These were not foci of inflammatory tissue but true lymph follicles. In some of these, which had become more rarefied in structure, fissures were found and these were becoming lined with flattened lymph cells. This, the author believes, explains the genesis of the larger cysts from this lymphoid tissue. Foci of epithelioid cells and a few giant cells were also found. The cysts contained a coagulated reticulum full of lymphocytes, the residue of lymph. An interesting point is the fact that some blood was removed with the fluid by aspiration, which might have led to the diagnosis of hemangioma. In view of the histologic findings, however, the diagnosis of true lymphangioma is certain, the blood having evidently been obtained from rupture of a blood vessel into the cyst. Nine cases of lymphangioma have been reported, and in none of these was the exophthalmos as extreme as in the author's case. Five drawings and a bibliography were included. S. R. G.

Schott, K. Socalled Coloboma of the Macula. Klin. M. f. Augenh., v. 67, 1921, p. 415.

Schott describes two cases of coloboma of the macula in sisters of 7 and 8 years. A brother of the father had the same affection, and three out of five children of the father's second brother were reported to have poor sight and nystagmus. In both cases, the visual fields were concentrically contracted for white and colors, no scotoma. The borders of the horizontal oval foci, of symmetric location were very sharp and intensely pigmented. The interior was, in one case, yellowish red, in the other white, and filled with irregular figures of pigment and remnants of the choroidal plexus. It was slightly excavated as shown by parallax. In the left eye of the younger sister, a larger retinal vessel coursed over the focus.

So far a family occurrence of coloboma has not been described. Perhaps this family congenital eye affection, which does not prove to be of hereditary nature, may be explained by Elschnig's theory of a localized proliferation of both strata of the secondary ocular vesicle at the posterior pole, an active process of the ectoderm thru which the normal development of the descendents of the mesoderm were damaged as the final malformation showed. C. Z.

Wachtler. Therapy of Vernal Conjunctivitis with Afenil. Klin. M. f. Augenh., 1921, v. 67, p. 446.

Encouraged by the striking case of vernal conjunctivitis treated by Cords with intravenous injections of 10 c.c. of 10% afenil (chlorid of calcium urea) Wachtler tried this in 3 cases. Only one could be observed for sufficient time, viz., a man aged 19, in whom the conjunctiva of the upper lid showed the characteristic papillary hypertrophy while the limbus presented no changes except increased injection. After 9 days the conjunctiva was almost smooth, and the distressing symptoms had entirely disappeared. C. Z.

Cramer, E. Thrombosis of Central Retinal Artery with Swelling of Disc after Influenza. Klin. M. f. Augenh., v. 66, 1921, p. 488.

A man, aged 47, lying ill with influenza, suddenly noticed a contraction of the visual field of his left eye, leading in 3 days to complete blindness. The optic disc was very much swollen, snow white, borders indiscernible, as the disc was surrounded by an extensive greyish white opacity of the retina. Vessels not different from those of the right eye, but on the disc of the opaque retina small irregular hemorrhages, and at the center a cherry red spot.

Cramer assumed an arteritic thrombosis of the central artery from the influenza infection, far behind the visible portion. Since, beyond the lamina cribrosa both vessels lie close to each other in a sheath, the secondary moderate thrombosis of the vein in connection with the pressure of the closed artery did not lead to the classical picture, but to a stasis evidenced by the

swelling of the disc. After 3 weeks, the disc was atrophic. Vision returned to counting fingers at 1 1/2 mm. excentrically. C. Z.

Sidler-Huguenin, H. Ocular Syphilis in Second Generation. Klin. M. f. Augenh., v. 66, 1921, p. 44.

In all, 36 hereditary luetic families with 65 children were so carefully examined that certainly nothing pathologic could escape. Their statistics are superior to former ones by the additional serologic and roentgenologic examinations. In 22 families with 48 children, the mother, and in 14 families with 17, the father was an hereditary syphilitic. In 36 marriages 14, i. e., 28% or 1/3 of all 50 marriages, remained without children. Thus the procreation in hereditary luetics is very considerably diminished. The male sexual gland is more damaged by hereditary lues than the female. In the 64 children examined, no changes characteristic of syphilis could be ascertained. In a few cases, dystrophies were observed, which, however, cannot be considered as specific, especially in children of poorly nourished families of laborers. The statistics were taken from 2/3 policlinic and 1/3 private patients. The author urges Wassermann test before every marriage, in order to institute energetic antiluetic treatment, if positive. If the man is an hereditary luetic, the chances for healthy children are greater. C. Z.

Kreiker, A. Microscopic Findings in Ocular Conjunctiva and Genesis of Pannus. Klin. M. f. Augenh., 1921, v. 67, p. 235.

Kreiker excised, in 11 cases of trachoma, pieces of ocular conjunctiva 8 to 10 mm. long and wide immediately above the limbus, and treated them according to the usual methods for microscopic examination. In two cases without pannus, these apparently normal pieces of conjunctiva revealed normal, smooth epithelium. Immediately under it was a firm plasma cellular infiltration 60 to 100 μ thick, not sharply defined toward the submucosa, somewhat accumulated around the vessels.

These cases showed that the process advances more or less gradually from the fornix. At a given moment the parts above the limbus are infiltrated but the cornea not reached, as the limbus on which the loose subepithelial tissue ceases arrests the progress. After breaking this obstruction, the cornea becomes involved and pannus develops. According to these observations, the point of attack lies in the connective tissue, not in the epithelium. C. Z.

Geis, F. Acute Parenchymatous Keratitis in Epidemic Parotitis. Klin. M. f. Augenh., 1921, v. 67, p. 67.

In contrast to other cases published, Geis saw in a girl, aged 10, affected with left sided epidemic parotitis, an acute isolated parenchymatous keratitis of the left eye, without participation of the uvea, which healed after 6 days. The formation of streaks, the equally concentric clearing from the periphery, and the sudden disappearance of the opacity indicated its origin from effusion fluid into the parenchyma, due to toxic infection, similar to that in influenza. C. Z.

Davids, H. Actinomycosis of the Cornea. Klin. M. f. Augenh., 1921, v. 67, p. 69.

In the center of the left cornea of a woman, aged 30, was a striking white spot of from 1 to 1.5 mm. in diameter, sharply defined, of finely granulated surface. Under the loupe it looked like a fine compressed blossom of 3 leaves. The lower half of the cornea was occupied by an adherent leucoma. The deposit was easily removed with a knife.

After treatment with hydrochloric acid, it consisted of typical glomerulus bodies in form of an actinic wreath, of homogeneous contents. There were no mycelia, and cultures did not grow, showing that these bodies were dead. The bodies must be regarded as a reactive product of the organism. The actinomyces was probably aerobic, as it presented no inclination to grow into depth.

The clinical picture of corneal actinomycosis is variable. It does not

lodge in the intact cornea, according to the experience in actinomycosis of the lacrimal canaliculi, but only after injuries of the cornea. In this case the corneal tissue, changed by disease, offered little resistance to its invasion.

C. Z.

Leslie Paton. Tabes and Optic Atrophy. British Journal of Ophth., 1922, vol. 6, p. 289.

On looking over a series of visual fields in tabetics, one cannot help being struck by the remarkable variability that they show, and the marked disproportion that exists in different cases between the loss of visual acuity and the loss of visual field. Is there anything in the underlying pathologic changes which will explain this variability?

The author discusses this question in great detail. A summary of his pathologic conclusions is that the toxin was always a purely hypothetic substance, evoked to explain degenerations taking place where no active organisms could be found. In the light of the work of Noguchi, Levaditi, Mott, Head, Fearnsides, McIntosh and Fildes, it must now be allowed that all the manifestations of syphilis are due to the local production of toxins in the presence of the spirochete, but that the reaction between the spirochetes and the tissue varies at different periods and in different tissues, either because of a diminution of the number and virulence of the spirochetes, or because of differences in the resisting power of the tissues to the action of the toxin. There still remains, however, the unsettled question as to whether this locally produced toxin acts directly on nerve tissue, producing a parenchymatous degeneration, or primarily on connective, vascular and lymphatic tissues, with a consequent secondary nerve degeneration; or a third possibility, that the nerve degeneration and the connective tissue degenerations are coordinate results due to the presence of the spirochetes in both tissues.

Often, disturbance of dark adaptation, precedes the smallest obvious change in visual acuity, fields or color sense.

Uhthoff's, and also Stargardt's classifications of the visual fields are discussed. Eleven fields accompany the contribution, illustrating the various classifications. Eight of these are from the author's own work.

D. F. H.

Saint Martin, de. Pemphigus of the Conjunctiva and Heredosyphilis. Ann. d'Ocul., 1921, v. 158, p. 338–349.

After reviewing the history of conjunctival pemphigus and the theories concerning it, the author reports a case of a woman of 24, where the lesions first appeared at the age of 15 months. She had had several operations, probably for trichiasis. Her appearance was typical of heredosyphilis. Her eyes showed corneal and palpebral changes characteristic of pemphigus in different stages of evolution, more pronounced in spite of the typical pemphigus history. No other part of the body showed any lesion. The first treatment was to correct the position of the lid margins and to treat the trichiatic keratitis. A Wassermann was strongly positive, so, later, 40 intravenous injections of mercury cyanid were given, which were followed by 4 injections of neosalvarsan. This was followed by treatment with Gilbert's syrup. During the period of treatment there were no exacerbations, the general health improved and the local condition was satisfactory. Then the patient was not seen for 5 years, during which time the treatment was neglected. When seen again, there were exacerbations of the local condition, but these yielded to Gilbert's syrup and then became stationary.

C. L.

Bourguet. Operative Technic for Dacryocystitis without External Scar. Soc. de Méd. de Paris. Abst. Gaz. des Hôp., 1922, v. 95, p. 829.

The method has been used on 18 cases, with good results in 16. It consists in detachment of the nasal

mucosa of the ascending branch of the superior maxilla and both surfaces of the inferior turbinate, resection of the anterior half of this bone, and opening of the bony lacrimonasal canal at its inferior part. Opening from below upward of the entire internal part of the canal. Ablation of the entire internal wall of the membranous canal and of the sac, guided by a probe introduced thru the lacrimal passageway, suture of the detached nasal mucous membrane. C. L.

Holth, S. Microscopic Examination of Subconjunctival Scars after Sclerotomy for Glaucoma. Norsk. Magazin for Laegevidenskaben, vol. 82, p. 717.

The author reports the microscopic findings in the eyes of six cases of chronic glaucoma, which he had operated 5 mos. to 6 yrs. previous to death. In each case sections show actual fistulæ to be present. Some cases, too, showed the conjunctiva over the scleral defect to be very thin and, possibly, containing a secondary fistula. This thinning of the conjunctiva he attributes to extensive scar formation under the conjunctival flap, and therefore he advises that, in operating, the conjunctiva should be dissected away over as small an area as possible. D. L. T.

Guist, G. Reaction of Light and Dark Irides to Homatropin and Atropin. Wien. med. Woch., 1921, no. 24, p. 1055.

All light irides contain crypts and can be paralyzed with homatropin. The dark irides are only sparingly endowed with crypts, but dilatation is possible with homatropin. Dark irides without crypts do not react with homatropin but can be paralyzed only with atropin. H. A.

Hanke, V. Double Perforation of the Eyeball and the Traveling Retrobulbar Fragment of Iron. Wien. med. Woch., 1921, no. 24, p. 1058.

Hanke reported a case of a boy, in whom a small fragment of iron entered the cornea and posterior wall of the eyeball, and was arrested in the sheath of the optic nerve at its entrance into the sclera, and had in the course of a few weeks taken a lower position of several mm. and outward, and descended then downward toward the nose. The course of the splinter was observed by means of Roentgen pictures. H. A.

NEWS ITEMS

Personals and items of interest should be sent to Dr. Melville Black, 424 Metropolitan Building, Denver, Colorado. They should be sent in by the 25th of the month. The following gentlemen have consented to supply the news from their respective sections: Dr. Edmond E. Blaauw, Buffalo; Dr. H. Alexander Brown, San Francisco; Dr. V. A. Chapman, Milwaukee; Dr. Robert Fagin, Memphis; Dr. M. Feingold, New Orleans; Dr. Wm. F. Hardy, St. Louis; Dr. Geo. F. Keiper, LaFayette, Indiana; Dr. Geo. H. Kress, Los Angeles; Dr. W. H. Lowell, Boston; Dr. Pacheco Luna, Guatemala City, Central America; Dr. Wm. R. Murray, Minneapolis; Dr. G. Oram Ring, Philadelphia; Dr. Chas. P. Small, Chicago; Dr. John E. Virden, New York City; Dr. John O. McReynolds, Dallas, Texas; Dr. Edward F. Parker, Charleston, S. C.; Dr. Joseph C. McCool, Portland, Oregon; Dr. Richard C. Smith, Superior, Wis.; Dr. J. W. Kimberlin, Kansas City, Mo.; Dr. G. McD. Van Poole, Honolulu; Dr. E. B. Cayce, Nashville, Tenn.; Dr. Gaylord C. Hall, Louisville, Ky.; Dr. Edward D. LeCompte, Salt Lake City.

DEATHS.

Dr. Joseph J. Duncan, Jr., of Pittsburgh, died July 27th, aged fifty, at the Presbyterian Hospital.

Dr. George M. Gould, of Atlantic City, died at his home, after an illness of three hours from heart disease, August 8th, aged seventy-four. He occupied a very conspicuous position as an American ophthalmologist, and was specially noted for his writings on errors of refraction as the cause of migrain and other nervous disorders.

Dr. Frank Wilson Martin of Lansing, Michigan, died July 17th, aged sixty-three.

Dr. Juan Santos Fernandez, of Havana, Cuba, died August 6th.

PERSONALS.

Dr. Rene Rousseau has been appointed Chief of the Ophthalmic Clinic at Angers, to succeed Dr. Bichon, deceased.

Dr. Thomas A. Woodruff has resumed the practice of ophthalmology at New London, Conn.

Dr. Myles Standish has announced his retirement from active practice of ophthalmology.

Dr. Virgil Wescott of Chicago, son of Dr. Cassius Wescott, entered into partnership with his father September first.

Dr. A. Maitland Ramsey, of Glasgow, has been appointed President of the Ophthalmological Society of the United Kingdom, for 1922-23.

Dr. A. S. Percival, of Newcastle-on-Tyne, has been appointed President of the Section of Ophthalmology of the British Medical Association.

Dr. Komoto of Tokyo, Japan, retired March 22, 1922, as professor in the Imperial University of Tokyo. His successor is Dr. Ishiwaga.

Dr. L. Webster Fox, of Philadelphia, sailed in July for Scotland, where he will read a paper before the annual convention of the British Medical Association at Glasgow.

Dr. John C. Dye, formerly of Statesville, N. C., has sailed for Honolulu, where he will have charge of the eye department of the Tripler General Hospital.

Dr. O. E. Fink of Danville, Illinois, has been appointed visiting oculist and aurist to the Danville branch of the National Home for Disabled Volunteer Soldiers.

Dr. N. Bishop Harman, London, has been elected a corresponding member of the Société Nationale des Sciences Naturelles et Mathematique de Cherbourg in recognition of his researches into the origin of the facial musculature.

Dr. George W. Jean of Santa Barbara, California, was host at the May meeting of the New York Ophthalmological Society which was held at Delmonico's. This was the 492nd regular meeting of this society.

SOCIETIES.

The American Academy of Ophthalmology and Oto-Laryngology will hold its 27th annual meeting in Minneapolis and St. Paul, Sept. 19 to 23, inclusive. The regular program, which will begin on Tuesday morning, will be followed by the Section on Instruction on the 22nd and 23rd of September. Special fare rates on the certificate plan will be issued by the Railroads.

MISCELLANEOUS.

The New York Association for the Blind has been left $65,000 by the will of Mrs. Mary Kingsland.

The Unity Hospital, Brooklyn, has acquired ground for the purpose of erecting a modern institution devoted to the treatment of diseases of the eye, ear, nose and throat, and oral surgery. The building will be four stories high and equipped with modern facilities.

At the annual meeting of the Iowa Association for the Blind, at Binton, a corporation was established to be known as the Iowa Foundation for the Blind. The purpose of the foundation is chiefly to raise money to be lent to worthy young blind persons who wish to enter business for which they have been fitted but lack capital. *Jour. A. M. A., Aug. 5, 1922.*

At the meeting of the executive committee of the American Red Cross in June, the sum of $12,000 was made available for making capital loans to blinded exservice men during the fiscal year ending June 30, 1923. These

loans are for the totally or partially blinded soldiers who wish to establish themselves in a small business. *Jour. A. M. A., Aug. 12, 1922.*

The Wintersteiner Collection of 13,000 microscopic preparations of pathologic changes in the eye has been acquired by the St. Louis University thru the generosity of Mr. Charles Rebstock of St. Louis. This collection, which is said to be the most complete in Europe, will be utilized for graduate instruction in ophthalmology. *Jour. of the A. M. A., Aug. 19, 1922.*

The Eye, Ear, Nose and Throat Hospital, New Orleans, will move into a new building on Tulane avenue. When the new, four-story, fire proof building is completed, the accommodations will represent an outlay of $500,000. Donations from various people have made this possible, $100,000 being given by Isaac Delgado, $20,000 by A. C. Hutchinson, $6,000 by Thomy Lafon, the negro philanthropist, and $50,000 by Mrs. John Dibert. Dr. Henry Dickson Bruns is surgeon-in-chief. He succeeded the late Dr. DeRoaldes, founder of the institution.

The Eye Sight Conservation Council of America, New York City, is establishing a special mailing list for lecturers and writers interested in the subject of conservation of vision. Data and material will be prepared and issued periodically to persons whose names are on the special mailing list. There is need for lecturers who will appear before local organizations, such as Rotarian and Kiwanis Clubs, chambers of commerce, schools and colleges, to present the subject of conservation of vision. *Jour. A. M. A., Aug 26, 1922.*

The Mississippi Association for the Blind, permanent organization of which was effected August 4, at Jackson, plans to make a survey to determine the number of blind persons in the state and the cause of blindness in each case. A social worker will be provided to make interviews and ascertain individual needs of the blind. The organization hopes to wage an educational war for the prevention of blindness and the conservation of vision, to assist the state board of health in stamping out diseases that cause blindness, and to help enforce the laws on the care of babies' eyes. *Jour. A. M. A., Aug. 26, 1922.*

The first training course in eye conservation ever given for teachers under the auspices of an American university was opened recently at Columbia University, with a class of twenty-one. A feature of the course is an observation class composed of children from New York City schools, whose defects of vision have placed them in the sight-saving classes. Dr. Robert R. Irwin, supervisor of the sight-saving department of the public schools of Cleveland, is general director of the Columbia course. A committee, composed of representatives of city, state and national organizations, which is cooperating with Columbia in the arrangement of these courses is headed by Dr. Thomas J. Riley. *Jour. of the A. M. A., July 29, 1922.*

Current Literature

These are the titles of papers bearing on ophthalmology received in the past month. Later most of them will be noticed in Ophthalmic Literature. They are given in English, some modified to indicate more clearly their subjects. They are grouped under appropriate heads, and in each group arranged alphabetically, usually by the author's name in **heavy-face type.** The abbreviations mean: (Ill.) illustrated; (Pl.) plates; (Col. Pl.) colored plates. Abst. shows it is an abstract of the original article. (Bibl.) means bibliography and (Dis.) discussion published with a paper. Under repeated titles are given additional references to papers already noticed. To secure early mention, copies of papers or reprints should be sent to 217 Imperial Building, Denver, Colorado.

DIAGNOSIS.

Birkhaueser, R. Basis of test types. Klin. M. f. Augenh., 1922, v. 68, pp. 732-738.

Coppez, H. Electric lamp for diagnosis of diplopia. (1 ill.) Bull. de la Soc. Belge d'Opht. 1922, No. 45, pp. 24-26.

Kearney, J. A. Value of eye observations in fractures of skull and head injuries. New York State Jour. Med., 1922, v. 22, pp. 341-344.

Strebel. Test for acuteness of vision. Schweiz. med. Woch., 1922, v. 52, p. 456. Abst. Internatl. Med. and Surg. Survey, 1922, v. 4 (8a-23).

Repeated title. **Lauber, H.** (A. J. O., 1922, v. 5, p. 514.) Internatl. Med. and Surg. Survey, 1922, v. 4 (8a-21).

THERAPEUTICS.

Beaumont, W. M. Butyn versus cocain. Lancet, Aug. 5, 1922, p. 304.

Cassimatis. Therapeutic value of milk injections in ocular disease. Bull. Ophth. Soc. Egypt, 1921, p. 65.

Fernandez, R. Radium therapy in eye, ear, nose and throat work. Jour. Philippine Islands Med. Assn., 1922, v. 2, p. 116.

Harris, S. J. Standardizing use of constant current in ophthalmology. Amer. Jour. Electrother. and Radiol., 1922, v. 40, p. 143. Abst. Internatl. Med. and Surg. Survey, 1922, v. 4 (8a-11).

May, J. W. Ophthalmic therapeutics. Jour. Kansas Med. Soc., 1922, v. 22, pp. 229-234.

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Schwarzkopf, G. Optochin iontophoresis and ultraviolet light therapy. Zeit. f. Augenh., 1922, v. 48, pp. 77-89.

Triebenstein, O. Metallic silver in eye after instillation of electrocollargol. Klin. M. f. Augenh., 1922, v. 68, pp. 749-752.

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Wick, W. Fixation forceps in enucleation. (3 ill.) Klin. M. f. Augenh., 1922, v. 68, pp. 780-783.

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Best, F. Arch of heavens and related questions. Depth perception. Zent. f. d. g. Ophth. u. i. Grenz., 1922, v. 7, p. 449.

REFRACTION.

Brudzewski, K. Spectacles formerly and now. Polska Gaz. Lek., 1922, v. 1, pp. 351 and 376. Abst. Internatl. Med. and Surg. Survey, 1922, v. 4 (8a-28) (8a-29).

Dohme, B. Contact glass in correction of keratoconus. (bibl.) Zeit. f. Augenh., 1922, v. 48, pp. 106-112.

Elschnig, A. Myopia. Med. Klin., 1922, v. 18, pp. 683-685. Abst. J. A. M. A., 1922 v. 79, p. 417.

Harris, S. J. Correction of refraction without glasses. Amer. Jour. Electrother. and Radiol., 1922, v. 40, pp. 246-255.

Hubbard, C. H. Protest against nonrecognition of optical defects. Hahnemann Monthly, 1922, v. 57, pp. 470-472.

Mazzei, A. Size of retinal image and optical correction after removal of unilateral cataract. Arch. di Ottal., 1922, v. 29, p. 224.

Thomson, E. Estimation of refraction without a cycloplegic. Brit. Jour. Ophth., 1922, v. 6, p. 379.

OCULAR MOVEMENTS.

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Cords, R. The nystagmus problem. Münch. med. Woch., 1922, v. 69, p. 693.

Papillon, P. H., and **Lestoquoy, C.** Congenital familial nystagmus with albinism. Bull. Soc. de Pediat. de Paris, 1922, v. 4, p. 128. Abst. Internatl. Med. and Surg. Survey, 1922, v. 4 (8a-37).

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Young, G. Subconjunctival advancement. Brit. Jour. Ophth., 1922, v. 6, pp. 323-324.

Repeated titles. **Minkowski.** (A. J. O., 1922, v. 5, p. 515.) Arch. d'Opht., 1922, v. 39, pp. 441-444. **Muñoz Urra, F.** (A. J. O., 1922, v. 5, p. 597.) Internatl. Med. and Surg. Survey, 1922, v. 4 (8a-7).

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Addario La Ferla, G. Treatment of trachoma after method of Sculco. (2 ill.) Arch. di Ottal., 1922, v. 29, pp. 219-223.

Bakly. Streptothrix infection of conjunctiva. Bull. Ophth. Soc. of Egypt, 1921, pp. 49-53.

Best. Swimming bath conjunctivitis. Münch. med. Woch. April 28, 1922. Clin. Opht., 1922, v. 26, p. 402.

Demets. Chancre of conjunctiva. Bull. de la Soc. Belge d'Opht., 1922, No. 45, pp. 38-40.

Gemblath. Treatment of trachoma with subconjunctival injection of cyanid of mercury. Arch. d'Opht., 1922, v. 39, pp. 428-434.

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Wilson, A. K. Trachoma and conjunctival folliculosis. Florida Med. Assn. Jour., 1922, v. 8, p. 219.

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Duverger and Lampert. Superficial and keratitis profunda. Soc. Franç. d'Opht., 1922, May. Ann. d'Ocul., 1922, v. 159, p. 558.

Ganguly, S. K. Keratectasia. (2 ill.) Calcutta Med. Jour., 1921, v. 16, p. 93.

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Coppez, H. Fever in uveoparotitis. (2 ill. bibl.) Bull. de la Soc. Belge d'Opht., 1922, No. 45, pp. 40-48.

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